VOL. XLVI. No. 4 OCTOBER, 1952

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The BRITISH JOURNAL of TUBERCULOSIS and DISEASES OF THE CHEST

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BAILLIÈRE, TINDALL & COX, LTD.
7 & 8, HENRIETTA STREET, LONDON, W.C.2

Quarterly: single copies, 8/-. Annual Subscription, 30/- (U.S.A. 5)

The British Journal of Tuberculosis and Diseases of the Chest

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BRITISH JOURNAL OF
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EDITORIAL

THE past decade has seen important advances in many fields of tuberculosis. The surgeon has extended the scope of his work and our views on changing methods of treatment are still in a state of reorientation, while the new drugs are almost like an alchemist's dream. The outlook for the afflicted individual has improved, and the death rate has undergone a spectacular fall. We are dazzled by the work of a few, and we are immersed in mass investigations which we call research. Yet fundamentally we hardly know why the disease is there, why it waxes and wanes in the course of its downward trend in communities, why it strikes the individual at certain ages and in certain forms, sparing some organs and affecting others, and why his reaction to it cannot be foreseen with any degree of certainty. Much is taken for granted and much is explained by views which are superficial and uncritical. When this is so it is salutary to turn to the work of one who is comprehensive in outlook, scientific in approach, philosophical in discussion, judicial in appraisal of facts, rationalistic in the formulation of conclusions, and has vision which sees beyond the immediate gross problems of the individual and the community and seeks the underlying influences which are at work in both. It is probably true to say that no man in recent years has exercised a more profound influence on thoughtful students of tuberculosis than Arnold Rich, and the advent of a new edition of his work on the pathogenesis of tuberculosis* is something of an event.

The innate powers of resistance play an important rôle in diseases of bacterial origin, and in tuberculosis their rôle is probably dominant. How else can we explain the gap between the 80 per cent. or more who are infected, and the 1 per cent. or less who are diseased? Yet we find it difficult to dissociate this imponderable factor from environmental and social conditions which play such an important part in the spread of the disease, and which are inextricably bound up with the problem of community resistance. We owe to Rich a clearer understanding of that native resistance which is our heritage and of the acquired resistance which is built on it, and yet is not the same thing. It is these which function in our favour, but which are intensely sensitive to adverse circumstances which react unfavourably on body and mind; and it is these which come again into their own when environmental and economic conditions improve. Infection we always have and the balance can be both tipped in its favour and restored to its level.

* *The Pathogenesis of Tuberculosis*, by Arnold R. Rich, M.D., Baxley Professor of Pathology, The Johns Hopkins University School of Medicine. Blackwell Scientific Publications, Oxford. Pp. xxvii+1028. Price 107s. 6d.

When we think of infection in tuberculosis, are we always clear in our minds about its manifold problems as they affect the organism and the host? Do we know why some young children can live in infectious tuberculous households and remain tuberculin-negative and clinically well? Are we right in attributing to lack of resistance in the newly born child the high mortality in these early months of life? Do we know why we have a problem of young adult tuberculosis, why this mortality rise is not seen at these ages in some other countries, and why the danger is accentuated in the female? Have we any justification for talking loosely of degrees of virulence of the tubercle bacillus, and do we know what in fact constitutes virulence? Is it true that super-infection—that is, fresh infection when active infection is already there—is rare?

Do we have clearly defined views on the vexed question of allergy, or has the growing use of B.C.G. led us to look on cutaneous sensitivity to tuberculin as being synonymous with immunity to tuberculosis? We know well the dangers of the allergic response in the course of the disease and the rapid superficial improvement which its control by restriction of body movement, and consequent slowing of the respiratory and circulatory mechanisms, can effect. Is allergy wholly bad or does it play some part in tuberculosis resistance? The relation of hypersensitivity (a term which Rich prefers to allergy) to immunity is no mere academic question. It has engaged the minds of men like Krause and Willis and many other distinguished workers in tuberculosis, but few have stated their views with such clarity and cogency as Rich. Even if we do not accept his views in their entirety they leave with us a stimulus to thought and a better understanding of many clinical phenomena and problems.

Is the vexed question of exogenous and endogenous re-infection (*pace* the contradiction in terms of the latter) settled? Are we not inclined to be partisans in this matter and to keep our minds closed to the possibility that both are at work? It would indeed be strange if it were otherwise, and there is plenty of clinical evidence that they are. If the primary infection passes in logical sequence to the so-called reinfection types of tuberculosis, are we sure of links which are often far from obvious, and do theories which are valid in one group of countries hold in others which differ ethnically and are in a different phase of industrial development?

Finally do we know why the mortality from tuberculosis is declining and why there has been such a remarkable acceleration in the last two years? Do we not often weave into our explanations theories which have little foundation on fact, which reflect our own wishes or views and ignore little obvious but fundamental causes? The whole problem is intricate and composite, and it is difficult to unravel the closely related and often contending factors which are at work. The fight against tuberculosis will not be won by shibboleths or slogans. It will not be won by advances in surgery, and it is very unlikely that mass dosing with drugs will bring us much nearer its control. The individual worker probably credits himself with greater power and influence than are his—for he has little control over many social and environmental conditions—but he has a front-line part to play and he will play it all the better for a rational understanding of the manifold problems which confront him in the course of his work and which are fused into a great national problem.

There are none who will not learn from Rich's writing, and there are few who will not study this fascinating volume with a sense of personal humility. Even if the edition differs from its predecessor only by the little which is added to it, and if there are a few sections such as that on infection from milk which discusses conditions which are remote from modern practice, the work remains unique in its erudition, in its critical comprehensiveness, and in its philosophical and yet judicial analysis of the great problems of tuberculosis. Arnold Rich leaves us in his debt.

C. C.

GENERAL ARTICLES

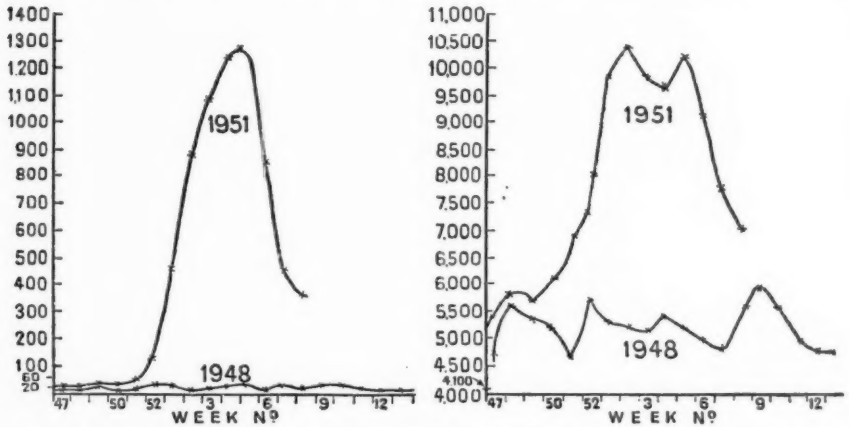
INFLUENZA

MISCELLANEOUS NOTES BY W. H. BRADLEY

INTRODUCTION

IN the Introduction to the Chief Medical Officer's report "On the State of the Public Health" for the year ending December 31, 1948, it is stated: "The outstanding epidemiological feature of 1948 was the absence of epidemic influenza."

The deaths ascribed to influenza in the 126 great towns of England and



These two charts are reproduced from Dr. Bradley's paper in the *Proceedings of the Royal Society of Medicine*, vol. 44, p. 789, by kind permission of the Editor.

Wales in the first quarter of the year numbered 280 only: a figure less than one-seventh of the average in the preceding ten years. There was, moreover, nothing to show that influenza virus was responsible for any of these deaths: in fact the virus reference laboratories of the Public Health Laboratory Service failed to recover virus from any of the material sent to them at the time. 1948 therefore provides a base line against which the virus A epidemic of 1951 can be compared. The following charts (Bradley, 1951) showing influenza deaths and total deaths give some idea of the damage done by an epidemic which was of moderate severity only (Chart). Logan (1951) estimated that in England and Wales some 50,000 people died directly or indirectly as the result of influenza during January and February 1951. Massey (1951) reported that there were 1,393,000 cases claiming sickness benefit among 22,700,000 insured persons during the same two months.

The nadir in 1948 emphasises the importance of influenza virus as the most lethal of the communicable infections in temperate zones; by comparison

tuberculosis, in all forms, has not killed 50,090 a year since the first decade of this century, and the deaths in 1951 will almost certainly number less than 16,000. The absence of influenza in 1948 also shows that the disease is not inevitable.

Influenza Virus

Any real knowledge about influenza virus began in 1933 with the epoch-making discovery by Smith, Andrews and Laidlaw that ferrets are susceptible to infection by intra-nasal inoculation of a filter-passing agent from cases of influenza and that all routes other than the intra-nasal are innocuous to ferrets.

Influenza viruses are of medium size. At the present time the group is divided into two distinct and immunologically unrelated serological types termed influenza "A" and influenza "B" respectively. Although further serological types probably exist, they have not yet been identified. Each of the two known types is represented by numeruos different strains which are in many respects similar but may differ one from another as regards certain properties. Of chief importance is the fact that individual strains of influenza "A" virus or of influenza "B" virus may be far from identical immunologically with other strains of the same serological type. Indeed sometimes immunological differences between individual strains are so great as to cause considerable difficulty in identification and classification, which is a long and tedious business. This in itself has justified the setting up by the World Health Organisation of a World Influenza Centre, now located at the National Institute of Medical Research, Mill Hill, under the direction of Dr. C. H. Andrewes.

Certain things are fairly clear. Strains of influenza "A" virus do not possess, so far as is known, any antigenic components in common with strains of influenza "B" virus; animals either actively or passively highly immunised to "A" strains are fully susceptible to infection with "B" strains and *vice versa*; hyper-immune sera which contain, in very high titre, antibodies against "A" strains fail to give specific anti-viral reactions in neutralisation, hæmagglutination inhibition or complement-fixation tests with "B" strains and *vice versa*.

The very distinctive immunological reaction which sometimes distinguishes different strains of "A" or "B" virus, and the absence of cross immunity, make the problem of immunisation in the control of the disease a very difficult one. For example, we know that the 1947 epidemic strain of virus "A" (F.M.1) was immunologically quite distinct from the viruses previously used to make the vaccines already available. It was in fact something we had never met during the influenza history which began in 1933. Existing vaccines would have been useless against it.

Matters of practical importance regarding influenza virus are:

(1) It can be adsorbed on to and eluted from, *inter alia*, chicken red cells. When adsorbed it causes the cells to agglutinate and is the basis of the Hirst test used in the serological diagnosis of influenza virus infections.

(2) When virus is brought into contact with a susceptible cell it apparently disappears. Immediately after invasion, extracts of infected cells show less infectivity than an equivalent quantity of virus added to comparable suspensions of ground-up normal cells. After a few hours the cell has disintegrated, partially or almost completely, and a brood of perhaps 50 to 500 fresh virus

particles has been set free and each of these is essentially a self-reproducing entity.

(3) The interference phenomenon. Burnet (1950) inoculated mice intracerebrally with a mixture of a neurotropic virus and a non-neurotropic strain. There was inhibition of the usual power of the neurotropic strain to kill all mice in five to seven days. This interference between viruses is a well-known phenomenon, but in the experiment quoted by Burnet the virus subsequently recovered from the mice shared some of the qualities of the two antecedent strains. This suggests that in some way the genetic material of two virus particles entering a cell can mingle and be rearranged. There is drama in the thought that when two unrelated viruses meet in a cell the whole course of history may be changed !

Pathogenesis

A remarkable thing about the histology of influenza is the limitation of the primary lesion at the basal membrane of the bronchial epithelium (Mulder, 1949). Further, Fazekas de St. Groth has shown that in experimental infections the multiplication of the virus is also limited to the cells lining the respiratory tract, and that there is no involvement of anything but the respiratory epithelium.

The surface character of the infection is noteworthy, and it seems that the toxic effects which accompany this relatively superficial damage are not due to any soluble toxin but are probably a measure of the cell damage. The damage to the respiratory membrane seems to make the patient particularly susceptible to secondary infection. During epidemics of relatively low virulence such as, for example, the virus "B" of the first quarter of 1950, the occasional rapidly fatal case may occur. Stuart-Harris believes that most of the mortality in healthy young people is due to secondary invasion, particularly by *Staphylococcus aureus*. The immediate impression is that influenza patients should be nursed under conditions designed to prevent cross-infection. This statement that staphylococcal pneumonia accounts largely for mortality relates only to the experience of some recent years. What really happened during the great pandemic of 1918-19 or in Merseyside in 1951, when mortality was greatly exalted, is unknown.

The Pandemic of 1918-19

It is interesting to review, in the light of what we now know, the 1918-19 experience when this historic scourge affected the well-being of millions of men and women and destroyed more human lives in a few months than did the European war which preceded it in five years. The Registrar-General's figures during the forty-six weeks from June 23, 1918 to May 10, 1919 attributed 151,446 deaths to this cause in England and Wales alone. Approximately three-quarters of these deaths occurred in the second of the three waves of the epidemic. In 1918 there were 3,129 deaths per million and in 1919, 1,170 deaths per million of the population of England and Wales. The first of the three waves as measured by mortality began in this country at the end of June, reached its zenith in the first half of July and fell by the end of that month; the second wave commenced in October and reached its summit in November,

remaining high for two or three weeks and falling slowly to the end of December. The third wave began at the beginning of February 1919, rose to its summit in the third week of February and fell slowly to the end of March.

The first wave occupied approximately six weeks with eight weeks' quiescence; the second, twelve weeks followed by four weeks' quiescence; and the third, eight weeks. Overall figures for the whole country regarding any epidemic are in many ways misleading, and there were considerable local variations in periodicity, incidence and case mortality. The pattern, in fact, was very little different from that described by Franklin Parsons in his report of the 1889-92 influenza, which led to the conclusions that the disease was imported into this country, that it followed the lines of human intercourse, and that it prevailed independently of season and climate. This is a point I wish to make; in my experience influenza has never moved so rapidly that one could not see it moving and one has some confidence in the assumption that it is spread from person to person.

During the 1918-19 epidemic it was assumed that the exalted virulence during the second and third waves was due to some variable host factor, but I am very doubtful about this. Contemporary reports suggest that there were clinical as well as epidemiological differences between the three waves.

The first wave was one of " the three-day fever " type, with rapid convalescence and low mortality. The chief nervous complications were psychical, and sleeplessness was common. The remarkable thing about it was that it attacked youth and tended to be hæmorrhagic.

The second wave, beginning in October, ushered in a much more severe form of the disease, and now " heliotrope cyanosis " cases began to appear associated with a high fatality. As Sir Herbert French pointed out, of a thousand individuals attacked in the autumn, about 800 suffered from the three-day fever of a somewhat unusual severity; the remaining 200 displayed a pulmonary complication, and of these 80 were moderately severe and 120 desperately ill. An overall case fatality of 6-8 per cent. resulted. Epistaxis was particularly common. Some cases had pulmonary involvement from the beginning, others in a few hours or days, frequently ending up with bronchopneumonia. Less frequently the disease appeared to be a virulent toxæmia which nowadays we should probably call shock.

The third wave had much the same character as the second.

The different stages of the pandemic did not occur at the same periods or with uniform sequence in different countries, which seems to point more to a variation in the causal agent than to an alteration in susceptibility or in the character of secondary infections. We have also to remember that the third wave had in it a component producing encephalitis lethargica. In fact, I can see few objections to a hypothesis that there were three different agents, probably viruses, producing pandemic disease in 1918-19. Andrewes (1949) suggests that a series of virus mutants appeared in 1918-19, and I believe this view to be supported by more recent events.

Recent Epidemics

The establishment by World Health Organisation of the World Influenza Centre has given a new view of the global epidemiology of influenza. All over

the world there are now observations posts sending information and strains of virus to Mill Hill. This has made it possible for us to watch what appears to be the movement of virus round the world.

Prior to 1947 there is little or no information, but during the past five years the quality of observation has improved rapidly.

1947 is a convenient year with which to begin a chronological account. It has been mentioned that the virus A isolated in that year was immunologically quite distinct from strains recovered previously—and of which P.R. 8 was fairly representative. The new "Virus A Prime," sometimes referred to as *F.M.*1, is now believed to have been first isolated by Burnet in Australia during April-June 1946; and by Francis in U.S.A. early in 1947. The disease first made its appearance in this country in January, and for the next few weeks many isolated outbreaks were seen, particularly in schools and military camps.

All strains of virus isolated were virus A prime. Virus B, which had been recovered frequently in 1946, was not found. "A prime" strains were also isolated in Sweden and Holland, yet the disease throughout Europe was relatively mild and could not be distinguished from virus B infections on clinical grounds. In spite of very bad weather, influenza did not reach epidemic proportions.

1948: The nadir of influenza deaths and the assumption that there was no epidemic influenza in U.K. in 1948 has already been mentioned.

1949: The absence of virus influenza in 1948 cleared the decks, as it were, for 1949, in which year the strains isolated in Europe were a compact, apparently homologous lot closely related to but not identical with F.M.1 (1947).

The W.H.O. World Influenza Centre was now in action for the first time, and Andrewes (1949) was able to study viruses from many centres on the Continent. The chronology of these outbreaks led him to be "pretty certain" that influenza spreads and that epidemics are not due to activation of endemic disease.

The 1949 epidemic appeared to begin in Northern Sardinia in late September 1948. It was noted soon after in Sicily, and rapidly spread all over continental Italy in November.

During December a virus identical with that isolated in Italy appeared in Switzerland, the Austrian Tyrol and France, where it entered the Haute Savoie about the 15th of the month. It spread along the eastern border of France between the 15th and 20th, when it jumped to most of the country, sparing only a few departments, but did not cross the Rhine. It seems it was not present in Paris until about the 20th, and the attack did not become appreciable until Christmas. A further fortnight passed before the incidence reached a maximum and remained there until the end of January. By January 14 almost the whole territory of France was involved in an epidemic affecting about 20 per cent. of the population. Some communities experienced attack rates of 40 per cent. or more. From France the epidemic passed through Belgium and was seen to enter Holland at Eindhoven on December 30. It appeared in Leyden seven days later. By the end of January, Bulgaria and Slovenia were involved, and there were foci in Finland and Turkey and the French Zone of Germany. Later foyers appeared in south-west Bavaria and the Saar, and the incidence increased considerably in southern Spain.

A most interesting feature is that in England and Wales influenza deaths showed no significant rise during the first four weeks of the year. They were, in fact, lower than in any year except 1948. Nevertheless infection had been introduced; it was suspected in Ross-on-Wye on January 1, but London appeared to be free, although Paris was having a fairly bad time. On January 6, two travellers who left Paris the previous day became ill in London, and strains of virus "A" were isolated from them. Another traveller who had returned from Switzerland to Broadstone on the Hampshire coast on January 14 was ill on the 15th; virus "A" was recovered from one of her contacts, who carried the disease to the nurses' quarters at an Oxford hospital. At the time seasonal catarrhs were naturally prevalent and were sometimes mistaken for virus influenza, but none of the several dozen pairs of serum received at the Virus Reference Laboratory of the Public Health Laboratory Service at Colindale showed any evidence of influenza virus infection before the middle of January. It was not until a month after the conjectured date of introduction of the virus into the country that deaths began to rise significantly from 64 in the fifth week through 94, 158 to 251 in the eighth week. Low as these figures are, and allowing that they contain a considerable proportion of deaths in the aged not due to the virus, they reflect a rate of dispersal which is by no means explosive. The usual experience in England was that a foyer appeared in a locality and then fizzled out before reaching epidemic proportions.

It is also interesting that in October of 1948 influenza occurred in the Gilbert and Ellice Islands (Isaacs, *Lancet*, 1950) and was due to the same virus as our "Italian" influenza. It followed the arrival of Chinese labourers from Hong Kong.

1950: In the first quarter of 1950 deaths numbered considerably less than half the aggregate in the same period in 1949 when virus A had reached mildly epidemic proportions in some localities. The virus laboratories were no less vigilant, yet virus "A" was not recovered on a single occasion, although there were numerous isolations of virus "B." Serological examinations also gave no consistent evidence of virus "A" infection. The same was true in Holland and France.

The American experience in 1950 presented a striking contrast to ours, because although there were some virus "B" isolations the main incidence of influenza, which was the highest during the previous 5 years, was clearly due to the virus "A" prime group.

Hilleman, Mason and Rogers (1950) made a comparison between the 1950 U.S. strains of virus A and strains recovered in previous years, including the F.M.1 which appeared both in this country and in America in 1947, and was antigenically so different from anything seen before. They reported that "the results of hæmagglutination inhibition and protection tests indicated that a number of strains of influenza A virus recovered in 1950 differed antigenically from those isolated in previous years. Over a number of years, the divergence in antigenic structure is so great that the earlier isolated strains elicit few protective antibodies against the newly isolated strains." It follows that in 1950 there was, in America, a virus A influenza of which we in this country had little or no experience.

The frequent recovery of virus B in the United Kingdom during the first quarter of 1950 has been mentioned, and it is interesting to record that in the previous year, when virus A was prevalent during the first 2 months, virus B was not isolated until March.

1951: The beginning of 1951 brought epidemic influenza to this country with devastating effect, particularly in the great cities of the north of England. Figures regarding incidence and mortality are mentioned in the introduction to this paper: the mortality in one week at Liverpool was higher than at the peak of the 1918-19 pandemic, albeit on this occasion the old and not the young died.

The background to the epidemic in the United Kingdom is as follows. First news of an outbreak in Europe came from Denmark in November 1950, and soon afterwards from Sweden and Norway. Subsequently it was learnt that in Norway the incidence had been highest in November, and that in Oslo the peak was passed well before Christmas.

" The early appearance of the disease in Scandinavia was not unexpected, as there had been a small outbreak of A prime near Stockholm in June 1950, and on several previous occasions an autumn epidemic had followed the occurrence of local outbreaks in the previous May or June in the same general area." (Andrewes, 1950, W.H.O.)

From the global point of view the 1950-51 influenza was mild and the high mortality in this country, particularly in Merseyside, exceptional and, so far, unexplained. On the one hand is the suggestion by Semple (1951) that the coincident very bad weather in Liverpool had much to do with it, and on the other the observation by Andrewes that the virus recovered in Liverpool differed from the one prevalent in other parts of the country.

Andrewes and Isaacs (1951) examined seventy strains of virus A isolated during the winter and distinguished two types which they designated the " Scandinavian " and the " Liverpool " respectively. Both were widely distributed throughout the world—one or other being dominant in certain zones. Because the same type was, as a rule, found in contiguous zones, and also because of a definite sequence in the appearance of the disease in these zones, there is a strong impression of geographical spread of each type across vast tracks of the world. The Scandinavian virus was concentrated in Sweden and Northern Europe and the Liverpool virus in the Mediterranean area, but both were found side by side in England, Ireland, Italy, South Africa and Australia.

Significant in 1951 was the unexplained high mortality in Merseyside. Virus isolated in Liverpool was antigenically indistinguishable from Liverpool type strains derived from zones where the disease was mild, and Wilson, Smith *et al.* (1951) have pointed out that " the key to the Liverpool episode must be in biological characteristics of the causative virus which are not reflected in its antigenic constitution." These authors also discuss biological variations in four strains of virus A isolated in London in 1951 and show differences which could be due to mutation (*ibid.*, 1951). They say: " Variations of this type occurring in the course of epidemic transmission from man to man may well have been the crucial factor in the development of the 1918-19 pandemic and the severity of the Liverpool outbreak in 1951."

1952: Up to the end of May there was no evidence of epidemic influenza in Europe and also, more significantly, virus A, so widespread a year previously, was not recovered either in Europe or North America, although in both places special arrangements had been made for virus isolations. A few strictly localised outbreaks of virus B occurred in March and April. In England and Wales the deaths certified as due to influenza ranged between 25 and 45 a week in 160 great towns of the country. Although these figures are a little higher than in 1948 they still do not indicate that these deaths have been due to virus influenza.

The effect of the absence of the epidemic on the productivity of the nation is remarkable. In round figures, and by comparison with 1951, somewhere about a million fewer people lost work in January and February 1952. There were also 50,000 fewer deaths in the eight-week period.

Résumé of Recent Experience

From the above account it will be seen that the "odd" numbered years have had an epidemic content due to virus A and that the "even" years have been either free from influenza or have had disease due to virus B. When virus A has occurred here, it has been preceded by the observation of apparent movement of virus A infection into this country from other parts of the world. The impression is that virus A is imported. On the other hand, virus B behaves in a fashion more suggestive of an endemic infection. Almost every year since investigations began there have been isolations of virus B and, characteristically, the disease occurs in relatively localised community outbreaks involving perhaps one town and missing another. Apart from this difference in "epidemnicity" it can no longer be assumed that in any given cases a virus A infection will be more severe than a virus B infection, but there remains a strong suspicion that the roving virus mutates more rapidly than the static one. It is still a matter for conjecture as to whether there is a small steady change in the antigenic structure of strains of influenza as they travel around or whether, in fact, a new virus emerges and sweeps the world until man has taken its measure and established an immunity. In this connection the global epidemiology of influenza is going to be an increasing and perhaps a very important study. Already it has shown us the very obvious probability that the serious epidemics are imported, which means that, hypothetically, they could be prevented if we knew how to keep them out.

Possibilities of Prevention

The problems of immunisation against influenza are great. Yet it must be admitted that vaccine prophylaxis offers the greatest hope of success and studies are now being actively pursued by a committee of the Medical Research Council. Apart from immunisation, is there anything else which can be done? In this connection I would like to mention a few personal experiences. In 1949 Dudgeon and myself observed an outbreak of virus A influenza at Westminster School. The strain of virus involved passed readily into eggs, which made it possible to take random samples of the school population for the presence of virus. Unexpectedly we found that virus was recovered only

from sick boys. In 1950 Dr. T. Sommerville, while investigating an outbreak of virus B at Winchester College, recovered virus on one occasion from the garglings of a boy before he developed clinical signs of influenza. Even if allowance is made for this possibility that a patient may be infective immediately before the development of clinical signs, I am still of the opinion that the sick person is the main source of infection.

In 1947 Professor Stuart-Harris and myself made a rapid survey of the preparatory schools at Seaford at a time when there was a high incidence of virus A there. We saw one very surprising thing. In one street there were three residential schools in a group with less than 100 yards between them. The two boys' schools at the ends of this row suffered attack rates of 60 to 70 per cent., but the girls' school in the middle escaped entirely: there could be little doubt about this, because the girls' temperatures were taken night and morning as a routine and were carefully recorded.

Even in a bad influenza year one can always find semi-isolated communities which do not suffer. Presumably this is because the virus was never introduced. With the above observations in mind I think it not unreasonable to attempt to isolate the first cases of any new influenza-like disease in any community, and I think we should be prepared to extend this to the home. Although we shall never be able to measure the result, there is a very definite probability that the relatively simple measures of withdrawal and isolation of first cases might have a marked effect in preventing or delaying the spread of influenza. Just in the same way as the disease did not cross the Rhine in 1949, it may be that with a little more care we might learn how to prevent it from crossing the threshold of our own homes.

Summary

These notes call attention:

1. To the essential limitation of the primary lesion and of the virus of influenza to the columnar cells of the bronchial epithelium.
2. To the sick person as the main source of spread.
3. To the epidemiological pattern implying importation of infection into continents, countries, communities and the family.
4. To the fact that influenza is not inevitable, and
5. To the possibilities of prevention.

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THE DUTCH STUDENTS' SANATORIUM AT LAREN, HOLLAND

By F. N. Sickenga
Medical Director

The Dutch Students' Sanatorium, formally opened at the end of June 1952, has been partially functioning since November 1951, and is an extension of work begun in 1947 with fifteen patients. The sanatorium is situated sixteen miles from Amsterdam and fourteen miles from Utrecht, both towns with large universities.

An ordinary sanatorium of 330 beds for pulmonary tuberculosis and one of 250 beds for tuberculosis of the bones and joints are in the immediate vicinity and there is close collaboration with these institutions, especially the former, which is equipped with a theatre for major chest surgery and possesses facilities for the investigation of respiratory function. Reduplication of such equipment in the Students' Sanatorium would not be an economic proposition.

The Government guaranteed three-sevenths of the cost of the building, which was about 2 million guilder (£200,000), and the remainder was provided by private loans and gifts, including a substantial contribution from the Dutch West Indies.

It has 85 beds, 62 for men and 23 for women, divided into nine single-bedded rooms, thirty two-bedded rooms and four four-bedded wards. There is a large hall for concerts, plays and cinema shows with platforms at the sides for chaises-longues. The cinema is connected with the broadcasting relay system so that records and lectures can also be relayed. A room is set aside for Protestant and Roman Catholic services. There is also a library of some ten thousand books, a reading room, a smaller lecture room and a room for arts and crafts.

The conception of a students' sanatorium was evolved in Switzerland as far back as 1922 by Dr. Vauthier, who is still its director. The example was followed in 1933 in France and after the war in Belgium and Holland, where the initiative was taken by three University professors. One of them, Dr. F. C. Heringa, Professor of Histology at the University of Amsterdam, was from the outset chairman of the Executive Committee and is still President of the Board. In England and in Italy plans are understood to be in the making, and Indonesia, India, Turkey and Japan have similar schemes. Poland and Czechoslovakia are reported to possess students' sanatoria which the Iron Curtain has concealed from our view.

The aim is to provide suitable surroundings for the student during his treatment and to combine that treatment, as far as is medically justifiable, with the continuation of his professional studies.

In this connection I want to emphasise two points: the first is that the medical limits to professional studies must be well-defined and not vague. In frankly active cases studies are completely prohibited and in the less active

ones are confined to one or at most two hours a day. Only with the gradual diminution of bed-rest are the hours of study correspondingly extended. The second point is as follows: I use the term professional studies deliberately and in contradistinction to amateurism, for the case for professional studies during treatment has been a subject of considerable controversy in Holland. It has been argued that the ideal psychological state for a tuberculosis cure is a carefree holiday mood. One can subscribe to this view, but the difficulty remains that such a mood does not as a rule last longer than a few weeks and is certainly unlikely to persist for a year or more. Moreover, people with a settled position in life, without financial and other worries, can indulge more easily in amateur studies than young people whose mental development is at its peak and who still have to fight to establish their talents, abilities and careers. Prolonged sickness during this period of development has its special psychological dangers. Enforced bed-rest leads to an accumulation of energy that not seldom tries to find an outlet in different forms of mischief. Alternatively, the mind may regress to certain childish automatisms. The psychology of the tuberculous patient has recently received much more attention than formerly. In England and Canada Dr. E. Wittkower has studied this approach intensively. In Holland Dr. van Helsdingen, who is consulting psychiatrist to this institution, has written an excellent book on the subject. He believes that some of the psychological difficulties that may arise are not caused by the illness but by the treatment.

We here are convinced of the essential value of prolonged bed-rest in the healing of pulmonary tuberculosis, to say nothing of surgical tuberculosis. Indeed, as recent statistics have shown, some 60 per cent. of cavities can close with conservative treatment consisting of strict bed-rest combined with antibiotics.

In such circumstances everything possible must be done to ensure that these healing tendencies are not disturbed by the patient's own restlessness. If his mental energy is directed to professional studies it finds, at least partially, a natural outlet, and he is encouraged by the idea that he is not altogether wasting his time and is not a useless member of society.

A second question then arises: if his studies have reached the point where he is ready for an examination, can this additional stress be allowed? He himself will ask for it, since it is the tangible proof of his progress. In fact we may probably regard the passing of an examination as a kind of stimulant therapy with all its advantages and drawbacks—it may produce a tonic effect, but only if the stimulus is not too strong. Accordingly, we advise patients not in any way to hurry their studies nor to seek a preliminary examination before they are quite certain of their knowledge of the subject in question. We do this, moreover, in order to avoid as far as possible an awkward dilemma for the examiners whose feelings of compassion might lead them to lower their usual standards. It is a great help for the patient to be allowed to pass a preliminary examination in one subject of the curriculum at a time and, in actual fact, most teachers do not object to this arrangement.

Another problem is the attitude to adopt if the patient is not study-minded, for it has been suggested that the student who does not resume his studies when these are medically permissible has no place in a students' sanatorium.

This is not, I think, the right point of view, especially during the first part of the cure when bed-rest is indicated. At this stage the healing of the tuberculous lesion is the dominant consideration and the psychological equilibrium of the patient must not be disturbed by demands on him to study when he does not feel so disposed. If he prefers amateur studies or some handicraft, he must be allowed to pursue such interests.

In the second part of the cure, however, when the patient is gradually returning to activity and adaptation to his normal life is in sight, there is justification for a confidential talk with him about his future and for a decision as to whether he should complete his former studies, change them for another branch, or, in exceptional cases, abandon them altogether.

This work falls to the head of the educational department after a thorough discussion of the case at the staff meeting. His rôle in a students' sanatorium is important. He is, so to speak, the liaison officer between the university teachers and the students; he organises conferences and lessons which may be given in person or over the wireless; he organises the entertainments, such as theatre, cinema or concert (as a rule once in three weeks); he fixes the times of the religious services; and he is head of the sanatorium library. He also supervises the "arts and crafts" department, the example of Adrian Hill being followed in Holland.

I will not go into the medical treatment, since this differs little from that of other Dutch sanatoria. Let me only say that as far as collapse therapy is concerned we are more conservative than in England, while with regard to surgical therapy, especially segmental resections and lobectomies, perhaps a little more progressive. As regards clinical investigations, we attach great importance to bronchoscopy, bronchography, spirometry and, if need be, bronchospirometry. Negative sputa are cultured every one or two months; if the patient does not expectorate, gastric washings are investigated.

The problem of after-care for students has, to our mind, not yet been satisfactorily solved. In France this aspect of the care of the tuberculous student is much further developed. At Grenoble, Strasbourg and Paris are three after-care institutions where the students, after leaving the sanatoria, are still under medical supervision while continuing their university studies. The problem of after-care is particularly pressing for the many students who provide a high proportion of their study expenses by doing all kinds of other work (in Amsterdam these comprised some 25 per cent.). When such a student leaves the sanatorium a combination of these activities is no longer advisable and he has to choose between giving up either his job or his studies. Without the help of a scholarship or grant only the alternative remains.

A final word on the financial aspect. In all Dutch universities a University Health Service insures its members against a part of the cost of sanatorium treatment. Unfortunately this covers not more than one-half to two-thirds of the real cost, so that the remainder has to be provided by the parents or from other sources. Here difficulties frequently arise and it is essential that the insurance premium and benefits should be raised. A subsidy from the Ministry of Education helps with, but does not cover the cost of, the educational side.

A students' sanatorium has its special sphere and its special problems which

differ somewhat from those of an ordinary sanatorium. The medical and educational services must be dove-tailed and some place set aside for recreation. Individual desires of some patients have to be subordinated to collective rules, but restrictions should be no more than are required for sound discipline in the sanatorium. Frequent staff meetings between the Medical Director, his second-in-command, the head of the educational department, the matron and the occupational therapist are very useful. The general management, as well as the special problems of individual patients should be discussed at such meetings. We are constantly seeking the best solution for our many problems—medical, educational, sociological, technical and, alas! financial, for we believe that the idea of a students' sanatorium is sound and worth-while.

POTT'S DISEASE IN SOUTH AFRICAN BANTU CHILDREN

AN ANALYSIS OF RESULTS AND COMPARISON WITH LANCASHIRE FIGURES

By C. J. Kaplan, Durban, South Africa

From the C.S.I.R. Research Unit in Tuberculosis, Umlazi and King George V Hospitals

The great and growing problem of tuberculous infection in all its manifestations among the Bantu peoples of South Africa has its roots in malnutrition, poor housing conditions and overcrowding, is nurtured by ignorance and superstition among the afflicted and its eradication is hindered by inadequate facilities for treatment, although much has been done in this field during recent years. Skeletal tuberculosis takes its toll, especially among the children, and here as in other countries the spine bears the brunt of the disease. The reason for this predominance is not immediately apparent, although Frazer (1929) has suggested it is due to retrograde lymphatic spread from the cisterna chyli and thoracic duct against which these structures lie. Burke (1950) concludes that most cases of Pott's disease are due to lymphatic dissemination; he considers that a pulmonary focus spreads to cause a pleuritis from which bacilli are carried to the para-aortic glands, these glands undergoing caseation and the tuberculous process spreading to adjacent vertebræ either by contiguity or the lymphatic route.

Clinical Material

The Umlazi Mission Hospital is an open-air hospital for Bantu children ten miles from Durban. One hundred beds are occupied by cases of skeletal tuberculosis, of which ninety-two at present have Pott's disease. During the years 1945 to 1951 fifty-eight cases with Pott's disease were discharged. The clinical records and X-ray films of this series have been examined in order to assess the immediate success or otherwise of conservative treatment. Unfortunately a long-term follow-up of cases has proved impossible, since most of the children concerned have returned to the oblivion of the native reserves.

In order to have some basis for comparison the figures given by Dobson (1951) have been used, his 0-10 years age group being abstracted for this purpose. These will be referred to as the Wrightington series.

Sex and Age Incidence

In the Umlazi series the disease occurred thirty-nine times in males and nineteen times in females. This does not indicate any special predilection for males, as of the ninety-two cases of spinal disease at present under treatment in this hospital there are forty-seven boys and forty-five girls. All cases were under 13 years of age on admission, the majority between 4 and 8 years, as shown in Fig. 1, and thus allowing a fair comparison with Dobson's figures.

The Lesion

The cervico-dorsal spine was involved in eight cases (14%), the dorsal spine in eighteen cases (31%), the lumbo-dorsal in seventeen cases (20%), the lumbar spine in eleven cases (19%) and the lumbo-sacral in four cases (7%). This is shown in Fig. 2.

A comparison with the Wrightington series is shown in Table I. It will be seen that the variation in the siting of the lesion is considerable. The total frequency of vertebral involvement is shown in Fig. 3. The heaviest incidence is seen to be between the tenth dorsal and the third lumbar vertebræ.

Table I.—Incidence of Disease at Different Levels. Comparison of Umlazi and Wrightington Figures

Level	*Umlazi 58 cases* *Per cent.*	*Wrightington 138 cases* *Per cent.*
Cervico-dorsal	14	7
Dorsal	31	45
Lumbo-dorsal	29	2
Lumbar	19	33
Lumbo-sacral	7	3
	100	100

Careful examination of all radiographic films revealed that where the first X-ray had been taken before gross destruction of bodies had occurred, the primary bony focus was at an anterior edge of one or more vertebræ and that the first sign was a narrowing of the intervertebral disc space. This agrees with the findings in the Wrightington series.

The extent of the lesion was assessed by counting the bodies destroyed or diseased in each case. It was found that two, three or four bodies were involved most commonly, but eighteen cases had five or more vertebræ affected and one had nine. This frequency of involvement is shown in Fig. 4.

Complications

In this series pulmonary infection, paraplegia, abscess and glandular involvement topped the list of complications. The frequency of complications is shown in Table II. There were fourteen cases with pulmonary tuberculosis,

Table II.—Complications

Site	*P.T.B.*	*Para-plegia*	*Menin-gitis*	*Abscess*	*Sinus*	*Gland-ular*	*Skeletal T.B.*	*Skeletal Non-T.B.*	*Died*
Cervico-dorsal	1	5	0	0	0	2	0	2	0
Dorsal	4	8	1	5	1	4	1	1	3
Lumbo-dorsal	4	1	1	6	5	3	:	1	2
Lumbar	3	0	0	5	2	2	3	0	2
Lumbo-sacral	2	0	0	1	1	1	1	0	0
Total	14	14	2	17	9	12	6	4	7
Percentage	24	24	3	29	15	21	10	8	12

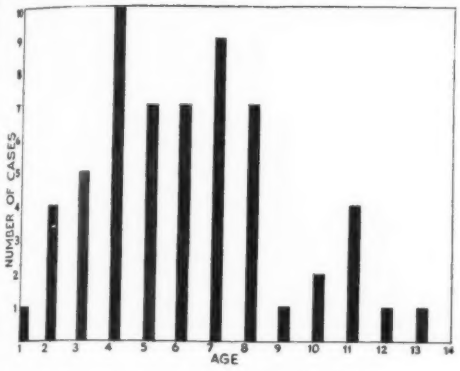

FIG. 1.—Age incidence on admission.

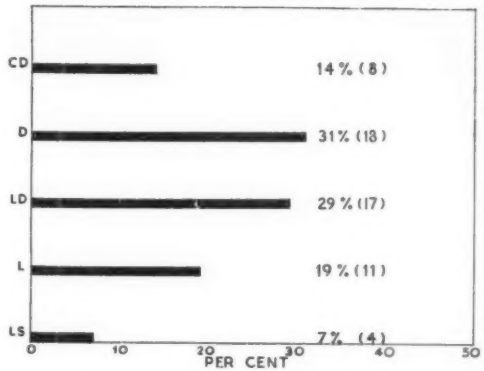

FIG. 2.—Incidence of disease by site.

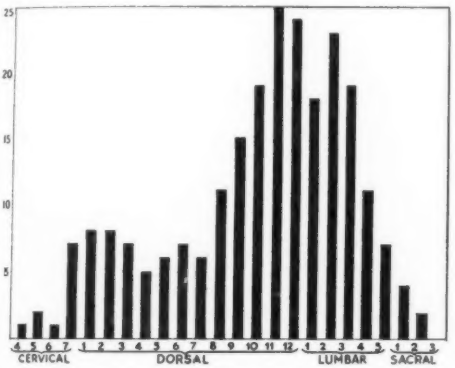

FIG. 3.—Total frequency of vertebral involvement.

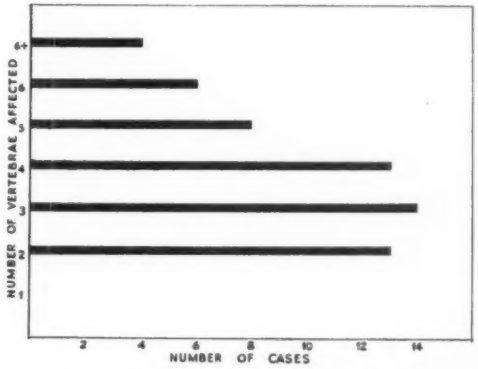

FIG. 4.—Frequency of vertebral involvement per case.

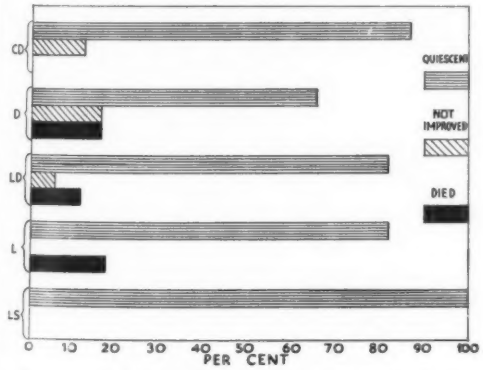

FIG. 5.—Condition on discharge by site of lesion.

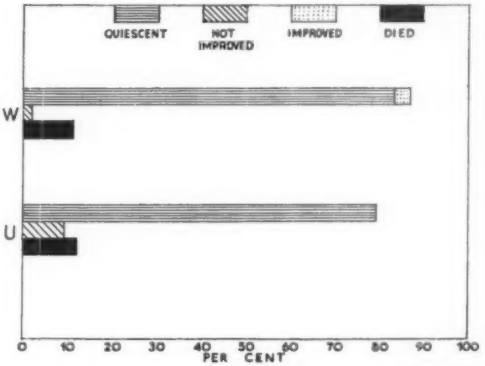

FIG. 6.—Similarity of results between Wrightington and Umlazi series.

To face p. 210

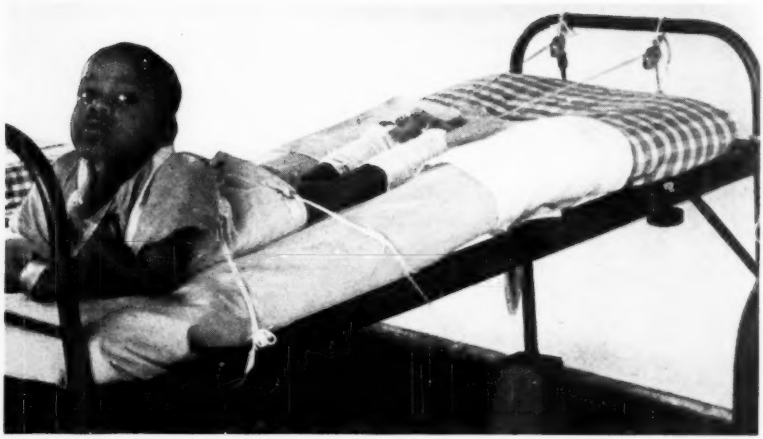

FIG. 7.—Prone nursing with excellent development of compensatory curves.

FIG. 8.—Thomas' posterior spinal support when disease is quiescent.

of whom one case died. There were fourteen cases of paraplegia (24%) as compared with 8% in the Wrightington series; their fate is indicated in Table III. Meningitis occurred in two cases, both of whom died. An abscess was

TABLE III.—RESULTS OF PARAPLEGIA

Level	*Recovered*	*Improved*	*Not Improved*	*Died*	*Total*
Cervico-dorsal ..	3	1	1	0	5
Dorsal	2	2	2	2	8
Lumbo-dorsal ..	0	1	0	0	1
Total	5	4	3	2	14

present in seventeen cases (29%) and nine cases (15%) developed sinuses, of which six were healed prior to discharge from hospital, two of the remaining three being transferred to another hospital for further treatment. In one case a sinus developed into a fæcal fistula, but this responded to streptomycin and healed after 30 gm. Glandular involvement occurred in twelve cases (21%); of these six were mediastinal, two cervical and four tabes mesenterica. Other skeletal foci of tuberculosis occurred in six cases (10%), of which there was one case each of tarsal infection, dactylitis, elbow joint and knee joint and two cases of hip-joint involvement. There were four cases with non-tuberculous skeletal conditions; one dislocation of the right hip from spastic paraplegia, one bilateral hip dislocation from the same cause, one Klippel-Feil syndrome who had cervico-dorsal disease, and one with a congenital absence of the left femur who walked, ran and played football on a pylon when his spinal focus was pronounced healed.

CONDITION ON DISCHARGE

Disease was quiescent in forty-six cases (79%), while five cases (9%) who were not improved were transferred to another hospital.

There were seven deaths (12%) in the series, one of which was due to measles in a case already up and about in a brace, one due to typhoid fever with intestinal perforation, and one to pulmonary tuberculosis, two to meningitis and two to cardiac causes—viz., congestive failure and tuberculous pericarditis.

These figures are reflected in Table IV and shown as percentages in Fig. 5, while Fig. 6 indicates graphically the similarity between the two series.

TABLE IV.—CONDITION ON DISCHARGE IN RELATION TO SITE OF LESION

Site of Lesion	*Disease Quiescent*		*Not Improved Transferred*	*Died*	*Total*	*Per Cent.*
	Without Appliance	*Wearing Spinal Support*				
Cervico-dorsal ..	7	0	1	0	8	14
Dorsal	3	9	3	3	18	31
Lumbo-dorsal ..	4	10	1	2	17	29
Lumbar	4	5	0	2	11	19
Lumbo-sacral ..	3	1	0	0	4	7
Total	21	25	5	7	58	100
		46				
Umlazi percentage		79	9	12		
Wrightington percentage		87	2	11		

The Wrightington figures are given in the last line for comparison, the "Quiescent" and "Improved" results being added to give 87%.

Spinal Deformity

It was felt that the degree of spinal deformity was greater than that experienced in a similar age group in Britain, where cases tend to present for treatment earlier and are nursed on frames to ensure rest—enforced, uninterrupted and prolonged, leading to better control of the disease and less extensive vertebral involvement. The deformity usually, but not necessarily, reflects the extent of destruction of vertebral bodies. In order to establish some basis for future comparison the deformity was graded as follows: " Slight" indicates a deviation of up to 30 degrees from the erect spinal posture, " Moderate " between 30 and 60 degrees, and " Severe " between 60 and 90 degrees. " Gross " indicates more than 90 degrees deviation with marked shortening of the spinal column and crowding of the ribs. The assessment was made from examination of all X-ray films and from clinical photographs where available, and is shown in Table V.

Table V.—Distribution and Frequency of Deformity

Site of Lesion	*Slight*	*Moderate*	*Severe*	*Gross*	*Total*
Cervico-dorsal ..	5	2	1	0	8
Dorsal	5	7	4	2	18
Lumbo-dorsal ..	8	4	3	2	17
Lumbar	10	1	0	0	11
Lumbo-sacral ..	4	0	0	0	4
Total	32	14	8	4	58

Treatment

The establishment in which this series was treated is a mission hospital unable to afford expensive orthopædic appliances, with the result that all cases are nursed prone (Fig. 7) and tied to the bed with a canvas corset, while bilateral leg traction with 4 lb. on each leg is employed more to induce the children to lie still than for any other reason, except if paraplegia supervenes; the absence of renal complications may be attributable to this position. It is however noticed that the children enjoy bouncing up and down on their beds from *joie de vivre*, and this probably accounts for the extensive vertebral involvement as a result of mechanical ulceration of soft bone. Once the vertebræ are remineralised a support is fitted; originally a steel spinal brace was supplied but more recently the Thomas' posterior spinal support (Fig. 8) has been introduced as it is cheaper, stronger and more efficient.

Streptomycin was not generally available during the period when this series was treated and was used for sinuses only during the latter years, although it is now given routinely in doses of 1 gm. every third day in association with para-amino-salicylic-acid (P.A.S.), as described by the writer in a previous communication (Kaplan, 1951).

Duration of Stay in Hospital

Because of generally unsatisfactory home conditions cases were kept in hospital for longer than would be the case in other centres. This is indicated by the comparatively large number discharged without an appliance (see Table IV), it having been discarded as no longer necessary.

Table VI indicates the average stay in hospital with regard to the different levels of the lesion. The overall average stay was forty-five months.

TABLE VI.—AVERAGE STAY IN HOSPITAL

Site of Lesion	*Average Duration (Months)*
Cervico-dorsal ..	35
Dorsal	43½
Lumbo-dorsal ..	54
Lumbar	47
Lumbo-sacral ..	47½
Overall Average ..	45½

Less two cases totalling six weeks; one died and one taken away against advice.

Conclusions

A survey of fifty-eight cases of Pott's disease in Bantu children all coming from under-privileged homes shows that, despite the absence of frame or plaster shell nursing and with a fairly high incidence of complications, the immediate results compare very favourably with those treated in a well-equipped and staffed hospital in Lancashire (Fig. 6).

Unfortunately a long-term follow-up of cases has not been possible. It is felt that this would in any case be disappointing by reason of the unfortunate home conditions to which these children return.

Summary

1. The results of treatment in fifty-eight cases of Pott's disease in Bantu children are analysed and compared with a series examined in Lancashire.

2. The incidence of the disease at different levels is found to differ considerably from those pertaining in Lancashire, although the site of the primary focus in the vertebral body is the same.

3. Extensive involvement of the spinal column is found to be common.

4. The recovery rate was 79 per cent., while no improvement occurred in 9 per cent. and 12 per cent. died. The figures compare favourably with the Lancashire series.

5. An attempt has been made to assess the degree of spinal deformity.

6. Treatment is outlined and mention made of the long duration of stay in hospital for social reasons.

ACKNOWLEDGMENTS

I wish to thank Mr. R. C. J. Hill for allowing me to include in this survey cases that have been under his care, and Miss Joyce le Brun for her help in sorting out clinical records and X-ray films.

The cases reviewed were under the care of Dr. B. A. Dormer, Mr. A. L. Allen and Mr. Karl Nissen, and more recently Mr. R. C. J. Hill and myself.

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TUBERCULOSIS AND PURPURA

By Philip Ellman and J. H. P. Johnson
From the East Ham Chest Clinic, London

The association of purpura with tuberculosis has been studied with interest for many years. Willan's Treatise on Cutaneous Diseases (1801) notes that "purple spots occur in the last stages of Pulmonary Consumption as the immediate forerunners of death". Winternitz (1912), in an analysis of fifty-one cases of tuberculosis of the spleen, found two instances in which the skin showed evidence of purpura or ecchymoses.

Witts (1946) states that purpura and a hæmorrhagic tendency have "been noted in chronic septic infections, . . . and in tuberculosis, expecially the miliary form." Wintrobe (1946) also enumerates tuberculosis (especially miliary) as one of the recognised infective causes of thrombocytopenic purpura.

During the last fifty years, however, it has been established that purpura is not always the signal of impending death in advanced cases of pulmonary or miliary tuberculosis, but can occur in more benign and non-fatal cases.

We have had under observation a boy who developed a tuberculous pleural effusion at the age of thirteen, followed two years later by thrombocytopenic purpura which was successfully treated by splenectomy. After a further two years tuberculous cervical adenitis appeared, but this too subsided uneventfully and he remains well five years later.

As it seemed that there was a possible relationship between the two diseases in this case, and as he made such a good recovery, we have searched the literature in an attempt to find what factors predispose to the appearance of purpura in tuberculosis, whether any particular type of case is particularly susceptible and if a gloomy prognosis is justified in all cases.

Case Report

D. H., male (æt. 13 years). First seen by one of us (P. E.) in October 1943, when he had a left pleural effusion (lymphocytic exudate). After eleven months' sanatorium treatment the effusion had completely reabsorbed and X-ray of his chest showed no parenchymal involvement. The Mantoux test 1/10,000 was noted to be positive on 26.2.44.

In April 1945 he developed spontaneous bruises and petechial hæmorrhages on the trunk and arms and had severe, repeated epistaxes. At this time he gave a history of always having bruised easily. Examination of his blood showed a "secondary anæmia" and the platelet count was 50,000 per c.mm.

He was admitted to hospital after a severe epistaxis and the blood picture on 23.6.45 was as follows: RBC, 3·1 million per c.mm.; Hb, 45%; WBC, 7,000 per c.mm.; platelets, less than 1,500 per c.mm.; bleeding time, 8 minutes. He was given a transfusion of two pints of whole blood and thereafter his hæmoglobin remained about 90%, but the platelet count remained low repeatedly.

On 23.11.45 he was admitted to Leatherhead Emergency Hospital under one of us (P. E.). On admission no petechiæ were seen and the spleen was not

palpable. The blood picture was as follows: RBC, 5·36 million per c.mm.; platelets, 11,000 per c.mm.; bleeding time, 8½ minutes and 20 minutes on different days.

Splenectomy was decided upon and was performed by Mr. Harold Edwards. The following day multiple petechiæ appeared around the eyes and under the conjunctivæ. A week later the hæmatological findings were: RBC, 4·7 million per c.mm.; Hb, 90%; platelets, 51,000 per c.mm.; bleeding time, 5½ minutes; clotting time, 6½ minutes. Two months later the findings were: RBC, 5·34 million per c.mm.; Hb, 87%; WBC, 10,600 per c.mm.; polys, 65%; lymphos, 36%; large hyalines, 3·5%; eosinophils, 0·5%; platelets, 170,000 per c.mm. Sixteen months later the findings were almost identical. His recovery was uneventful and no further hæmorrhagic manifestations have been noted to date, the blood picture now being quite normal.

The spleen was thought to be slightly enlarged and was reported on as follows: " Sections of the spleen show a histological picture as is seen in thrombocytopenic purpura. The follicles are prominent although not so very active in this spleen; but the sinuses are empty and the endothelial lining cells are prominent and active. There were no megakaryocytes seen."

In November he developed a mass of glands in the left supraclavicular region. These were clinically of a tuberculous nature and he was admitted to the Royal Sea Bathing Hospital, where he was given a course of X-ray treatment for the glands and a sanatorium régime over a period of two years. The glands subsided unventfully and no other evidence of active tuberculosis was found, serial X-rays of his chest always being negative. He has remained quite well subsequently and is now working.

Review of the Literature

Following Kaznelson's demonstration of the prompt and dramatic increase in the number of platelets in the circulation following splenectomy in certain cases of thrombocytopenia (1916), attention became focused on the spleen as the organ probably responsible for their depletion in such cases. Splenectomy was performed more frequently and cases were reported in which the removed organ showed nodular tuberculosis or miliary seeding, thus calling attention to the possible rôle of tuberculosis in the ætiology of some cases of thrombocytopenic purpura. Several cases were then reported of purpura occurring with miliary tuberculosis, and it became accepted that this was practically the only form of tuberculosis which caused purpura.

Perusal of the literature, however, makes it clear that purpura occurs no less often with other forms of tuberculosis. Of seventy-seven reported cases, only eleven were of miliary tuberculosis and of these, one described by Gouley *et al.* (1949) and two reported by Pratsicas (1924) resembled more the type of atypical abdominal tuberculosis reported by Friend and Thackray (1952) and earlier described by Blair and Pagel (1947). One of Friend and Thackray's cases and a similar case reported by Lapp (1948) developed panhæmocytopenia with purpura. Ball *et al.* (1951) reported three cases of acute tuberculous septicæmia, a type of miliary tuberculosis, with leucopenia; two cases had purpuric symptoms, one with thrombocytopenia.

Twelve cases showed miliary seeding or scattered nodules in the spleen either as an isolated autopsy finding or following splenectomy (eight cases). In two examples reported by Solomon and Doran (1939) and by Elliott and

Turner (1951) the spleen removed at operation showed extensive caseation. In the former the spleen was grossly enlarged (1,240 grammes), the liver was palpable three finger-breadths below the costal margin and there was panhæmocytopenia. Following splenectomy the blood picture returned to normal and fever and toxæmia which had previously been present subsided. Although autopsy evidence is lacking in this case, it appears probable that it, too, was an example of atypical abdominal tuberculosis; X-ray of the chest was normal.

The largest group of purpura was associated with pulmonary tuberculosis, no less than thirty-eight cases being described. The case reported by Greengard (1931) came to autopsy and proved to have, in addition to tuberculous bronchopneumonia, ileo-colitis, peritonitis and miliary infiltration of a kidney. Wright-Smith's case also had Hodgkin's disease, which had infiltrated the lymphatic glands, spleen and bone marrow and which undoubtedly caused the purpura.

Of the remaining cases, two reported by Blasio and Scrocca (1935) and Lebon *et al.* (1937) were skin tuberculides, two described by Ponteva (1935) and Martin de la Vega *et al.* (1939) of tuberculous cervical adenitis, two published by Baize (1923) of primary tuberculosis, and two reported by Davis (1943) had only dry pleurisy.

Finally, three cases were described in which purpura occurred in close association with intradermal Mantoux tests. The first, reported by Wright and Boccal (1923), was in a boy aged thirteen years who was tested with 0·1 c.c. of 1/1,000 O.T. and developed a severe local reaction. Three days after the injection purpura appeared, first on the same arm, but rapidly spreading to cover the entire body. The blood picture was as follows: RBC, 4·8 million per c.mm.; Hb, 82%; WBC, 2,400 per c.mm.; polys, 29%; lymphos, 21%; monos, 15%; eosinophils, 29%; platelets, 170,000 per c.mm. The purpura settled in one week, but twelve months later when he was tested with 1/100 O.T. he again had a severe local reaction with petechiæ on the same arm and systemic upset. The other two cases reported by Gonzalez Batlle (1941) were in girls aged seven and eight years. In each case purpura followed the injection within forty-eight hours and was accompanied by constitutional upset; in one there was a blood eosinophilia and in the other generalised œdema.

In thirty-one cases the purpura was of the thrombocytopenic variety, associated in seven instances with anæmia or leucopenia or both (six cases). Thrombocytopenia was noted in five out of eleven cases of miliary tuberculosis, ten out of twelve with tuberculosis of the spleen, eleven out of thirty-eight with pulmonary tuberculosis, one case of cervical adenitis, one with acute tuberculous septicæmia, two of the three cases of atypical abdominal tuberculosis and one of the three cases in which purpura followed Mantoux tests.

Panhæmocytopenia was present in six cases, as noted above; one with pulmonary tuberculosis and Hodgkin's disease, two with tuberculosis of the spleen, one with acute tuberculous septicæmia, one with atypical abdominal tuberculosis and one of purpura following a Mantoux test. The two cases with tuberculosis of the spleen, reported by Solomon and Doran (1939) and Engelbreth-Holm (1938), also had enlarged livers; in addition one had ascites

and the other a pleural effusion. Splenectomy was done in both these cases and complete recovery from the purpura and tuberculosis followed. Splenectomy was performed in six other cases in which the only evidence of tuberculosis was the finding of tubercles in the removed organ. In sixteen other cases the spleen examined at autopsy proved to be infiltrated with tubercles and in another with Hodgkin's disease. The marrow was reported as having been examined in three cases with thrombocytopenia; in two there were miliary tubercles and in the third, infiltration with Hodgkin's tissue.

TABLE I.—CASES WITH THROMBOCYTOPENIC PURPURA

Tuberculous State	*Reported by*		*Number*	*Age and Sex*	*Deaths*	*Anæmia*	*Leucopenia*	*Palpable Spleen*	*Splenectomy*	*Remarks*
MILIARY TUBERCULOSIS (5)	Villa	1928	1	38 F	1	—	—	—	—	
	Greengard	1931	1	3 F	1	—	—	—	—	Mantoux −ve.
	Ureta	1943	1	11 F	1	—	—	—	—	Mantoux −ve.
	Reggiani	1948	1	19 F	1	—	—	—	—	
	Gouley *et al.*	1949	1	51 M	1	+	—	++	—	Miliary marrow.
TUBERCULOSIS OF SPLEEN (10)	Kellert	1931	1		—	—	—	—	+	Recovered.
	Engelbreth-Holm	1938	1	44 F	—	+	+	+	+	Previous hæm. tendency. Recovered.
	Solomon and Doran	1939	1	30 F	—	+	+	++	+	Albuminuria. Recovered.
	Weiner and Carter	1941	1	42 M	—	—	—	—	+	Recovered. Mantoux +ve.
	Lapp	1948	3	36, 19 and 25	3	—	—	—	—	
	Elliott and Turner	1951	3		—	—	—	—	+ (3)	Extensive caseation in one spleen. All with miliary.
PULMONARY TUBERCULOSIS (11)	Blasio and Scrocca	1935	1	M	1	—	—	—	—	
	Nitti	1936	8		—	—	—	—	—	
	Giraud *et al.*	1942	1	18 F	—	—	—	—	—	
	Wright-Smith	1943	1	38 M	1	+	+	—	—	Hodgkin's dis. in glands, spleen, and marrow.
TUBERCULOUS ADENITIS (1)	Martin de la Vega *et al.*	1939	1	18 M	—	—	—	—	—	
ACUTE TUBERCULOUS SEPTICÆMIA (1)	Ball *et al.*	1951	1	47 F	1	+	+	+	—	Miliary tubercles in spleen, liver and marrow.
ATYPICAL ABDOMINAL TUBERCULOSIS (2)	Lapp	1948	1	64 M	1	+	+	—	—	Caseous Abd. and mediast. glands. Nodular tuberc. liver and spleen.
	Friend and Thackray	1952	1	60 F	—	—	—	+	+	Occ. tubercles in spleen. Recovered.
MANTOUX TESTS (1)	Wright and Bocal	1937	1	13 M	—	+	+	—	—	
(31)	TOTAL		31		12	7	6	5	8	

TABLE II.—CASES WITH PURPURA OTHER THAN THROMBOCYTOPENIC

Tuberculous State	*Reported by*		*Number*	*Age and Sex*	*Deaths*	*Anæmia*	*Leucopenia*	*Palpable Spleen*	*Splenectomy*	*Remarks*
MILIARY TUBERCULOSIS (6)	Garin	1921	1		1	—	—	+	—	
	Pratsicas	1924	5	22	4	—	—	—	—	Two resembled attyp. abdom. tuberculosis.
				22	—	—	—	++	—	
				23		+	+	—	—	
						—	—	—	—	Purpura after authohæmo-therapy in another.
						—	—	—	—	
TUBERCULOSIS OF SPLEEN (2)	Winternitz	1912	2	1 M	2	—	—	—	—	Both had large livers and ascites.
				67 M		—	—	—	—	
PULMONARY TUBERCULOSIS (27)	Bauch	1916	3		—	—	—	—	—	All had nephritis and œdema.
	Greengard	1931	1	11 F	1	—	—	—	—	Mantoux —ve, ileo-colitis and peritonitis.
	Blasio and Scrocca	1935	2		—	—	—	—	—	One Schönlein-Henoch.
	Ponteva	1935	1	11 F	—	—	—	—	—	Pirquet —ve, Nephritis and eosinophilia 7%
	Houcke and Boury	1938	1		1	—	—	—	—	
	Loubeyre *et al.*	1938	1	51 M	—	—	—	—	—	
	Bassi and Nuti	1940	1	28 F	—	—	—	—	—	Mantoux —ve.
	Vidal *et al.*	1941	1	17 F	—	—	—	—	—	
	Perlman and Fox	1941	12		—	—	—	—	—	
	Davis	1943	3		—	—	—	—	—	
	Curti *et al.*	1948	1	20 M	1	—	—	—	—	Nephritis.
SKIN TUBERCULIDES (2)	Lebon *et al.*	1937	1		—	—	—	—	—	
	Blasio and Scrocca	1935	1		—	—	—	—	—	
ADENITIS (1)	Ponteva	1935	1		—	+	—	—	—	
PRIMARY TUBERCULOSIS (2)	Baize	1933	2		—	—	—	—	—	Both Mantoux +ve.
PLEURAL EFFUSION (2)	Davis	1943	2		—	—	—	—	—	
MANTOUX TESTS (2)	Gonzalez Batlle	1941	2	7 F	—	—	—	—	—	Eosinophilia in one, œdema in the other.
				8 F	—	+	—	—	—	
ACUTE TUBERCULOUS SEPTICÆMIA (1)	Ball *et al.*	1951	1	54 M	1	—	+	—	—	Leucopenia.
ATYPICAL ABDOMINAL TUBERCULOSIS (1)	Friend and Thackray	1952	1	41 M	1	+	—	+	—	Anæmia and leucopenia.
(46)	TOTAL		46		12	4	2	3	—	

The purpura conformed to the classical Schönlein-Henoch syndrome in five instances, four with pulmonary tuberculosis and one with tuberculous cervical adenitis. In nine other cases œdema or nephritis or both occurred,

and it is probable that these were also examples of anaphylactoid purpura. Of these nine cases two had miliary tuberculosis, one a tuberculous spleen, three pulmonary tuberculosis, one a skin tuberculide and two were connected with Mantoux tests.

In the remaining thirty-two cases there is no real indication as to the type of purpura, no platelet counts being published, and they were variously described as hæmorrhagic diathesis, purpura hæmorrhagica or simply as purpura.

A previous hæmorrhagic tendency was noted in two cases described by Engelbreth-Holm (1938) and Friend and Thackray (1952).

Death occurred in twenty-four cases; ten out of eleven with miliary tuberculosis, five out of twelve with tuberculosis of the spleen, five out of thirty-eight with pulmonary tuberculosis, the two cases with acute tuberculous septicæmia and two of the three with atypical abdominal tuberculosis. In each group thrombocytopenia was noted in approximately half the cases. Autopsy was performed in every case except two with pulmonary tuberculosis. Among the miliary cases three had large caseous masses in the abdomen involving the spleen in one and the kidneys in another, apart from the widespread miliary infiltration which was present in all. One of the cases of pulmonary tuberculosis reported by Greengard (1931) also showed tuberculous ileo-colitis, peritonitis and miliary nodules in the kidneys. The cases of acute tuberculous septicæmia showed multiple necrotic foci in the liver and spleen, easily visible to the naked eye, and numerous microscopic necrotic foci containing acid-fast bacilli in most other organs. In the cases of atypical abdominal tuberculosis autopsy revealed tuberculosis of the abdominal lymph nodes, liver and spleen; in one the mediastinal glands were also affected and in the other there were microscopic tubercles in the bone marrow.

Discussion

It is clear from the literature that where purpura occurs with tuberculosis there is an etiological relationship between the two conditions except in a few instances when a chance association cannot be excluded. With the noteworthy exception of bone and joint tuberculosis, all other varieties have produced purpura on occasion. Thrombocytopenic and anaphylactoid purpura occurred in different cases, but, as with purpura in general, there was a small degree of overlap in some.

Before discussing the ætiology of purpura in these cases it is well to appreciate that thrombocytopenia and anaphylactoid purpura may have a common basis. Witts (1946) states that " purpura is often classified into thrombocytopenic and non-thrombocytopenic forms, but there is so much overlap between these two that the distinction often falls to the ground.... In all the purpuras, whether thrombocytopenic or non-thrombocytopenic, the first stage of hæmostasis appears to be at fault, the capillaries do not retract properly when they are injured and the bleeding time is prolonged." This common capillary defect has been demonstrated by Macfarlane (1941).

Allergy can produce thrombocytopenic purpura as well as the anaphylactoid variety, as pointed out by Doan (1947) and demonstrated experimentally by several workers.

Tuberculo-protein undoubtedly acts as an allergen and produces purpura in some cases, as in those reported by Wright and Bocal (1937) and Gonzalez Batlle (1941), where purpura followed intradermal Mantoux tests, and in other cases where the purpura was of the anaphylactoid variety. The same allergic mechanism presumably caused depression of the bone marrow in the first case. Gairdner (1948) concludes that " the Schönlein-Henoch syndrome is due to a hypersensitivity reaction, where the antigen may be either bacterial or non-bacterial in origin," and Wintrobe (1946) enumerates the tubercle bacillus as a possible exciting cause of allergic purpura.

The reason why a tuberculous (or other) allergen produces purpura in some cases and not in others may lie in an already existing hæmorrhagic tendency. This was noted in a case described by Friend and Thackray (1952), one reported by Engelbreth-Holm (1938) and in our case. The state of tuberculous allergy as indicated by Mantoux tests does not seem to be relevant; the test was performed in nine cases and in ours; it was negative in five children and one adult. In the second case reported by Bassi and Nuti (1940), the Mantoux test was negative at first but became positive as the purpura subsided. In the vast numbers of B.C.G. vaccinations that are being done and where Mantoux tests are carried out shortly afterwards no cases of purpura have been reported in the world literature. At the East Ham Chest Clinic, where some three hundred vaccinations have been performed, we have not found any evidence of purpura; in twelve children repeated platelet counts were done during the six weeks following vaccination and no deviation from the normal was noted.

In the six cases where thrombocytopenia was but a facet of a profound depression of the blood-forming elements it seems probable that direct bone marrow involvement was responsible. In the two examples where the marrow was examined, infiltration with tubercles was found in one described by Ball *et al.* (1951) and with Hodgkin's tissue in that reported by Wright-Smith (1943). However, as splenectomy resulted in prompt recovery in two cases, hypersplenism or toxic depression from remote tuberculous foci could also probably operate in such cases. An allergic hypothesis is probable in the case of Wright and Bocal (1937), where anaphylactoid purpura followed a Mantoux test and thrombocytopenia and leucopenia were present as well. Marrow infiltration was also found in three other cases with only thrombocytopenia, all of whom had evidence of abdominal visceral tuberculosis with miliary spread.

Nine splenectomies were recorded, including our own case, and in all excepting the last miliary tubercles were found in the removed organ. In every case recovery from the purpura was complete and the platelets returned to normal. In seven cases there was no real evidence, except inferential, of tuberculosis elsewhere in the body. In the case reported by Solomon and Doran (1939) the patient was febrile and toxic, the spleen was grossly enlarged (1,240 grams) and had a large necrotic abscess besides numerous microscopic tubercles, the liver was enlarged three finger-breadths below the costal margin, and a left pleural effusion appeared in the course of the illness. Splenectomy in this case was not only followed by recovery from the purpura, but the toxæmia and abdominal symptoms also disappeared and the patient made a complete recovery. In such cases where the spleen is the major seat of tuber-

culous disease splenectomy would appear rational in spite of the risks, especially as these could be lessened by streptomycin. In our case the purpura was preceded two years earlier by a pleural effusion and followed two years later by cervical adenitis. The incidental finding of scattered tubercles in a spleen removed for thrombocytopenic purpura appears to be of little significance as none of the reported cases developed active tuberculosis subsequently. It is probable that such cases have fostered the belief that miliary tuberculosis and tuberculosis of the spleen are the chief causes of purpura in this disease.

Death occurred in less than one-third of the cases and seemed to be related more to the severity and extent of the tuberculous process than to the incidence or type of purpura. Thus nearly all the cases with miliary tuberculosis, a hitherto uniformly fatal disease, died. The fatalities among those with pulmonary tuberculosis were in patients with extensive progressive disease, but in the case reported by Curti *et al.* (1948) death was caused by progressive glomerulonephritis, and that described by Houcke and Boury (1938) had fatal hæmorrhages from the lungs and kidneys. Four deaths occurred among the cases of acute tuberculous septicæmia and atypical abdominal tuberculosis; in each case caseating lymph glands, visceral involvement and miliary spread were present. Of the five deaths in cases with tuberculosis of the spleen, the two reported by Winternitz (1912) were in a boy aged one year and a man aged sixty-seven; they both had enlarged livers and ascites and the boy also had an abdominal tumour and œdema of the leg. It is apparent that there was more than just tuberculosis of the spleen in these cases.

In our case thrombocytopenic purpura occurred in a boy with a previous tendency to spontaneous bruising at a time when the tuberculous process was quiescent but not arrested; recovery from the purpura followed splenectomy and the spleen proved to be free of tubercles on examination. The purpura may have occurred fortuitously, but it seems likely that it was precipitated by the tubercle bacillus acting as an allergen in a patient already sensitised and with a pre-existing hæmorrhagic tendency. Recovery following splenectomy implies hypersplenism, but marrow involvement cannot be excluded as the marrow was not examined.

Conclusions

It is apparent that tuberculosis causes purpura in some cases, although the association may be accidental in a few. All forms of tuberculosis may be responsible and not only miliary or other severe and fatal types. The incidence of purpura in a case of tuberculosis does not always presage a fatal outcome, the prognosis depending on the severity and extent of the tuberculous process and being no different from similar cases without purpura. Cases with miliary tuberculosis and purpura have always died hitherto, as did miliary cases in general, but streptomycin and P.A.S. should halt their progress in the future.

The incidental finding of tubercles in a spleen removed for thrombocytopenic purpura is of little significance when no other evidence of tuberculosis is found, but such cases should be followed closely as a precautionary measure.

Tuberculosis causes purpura in various ways; in some cases allergy to tuberculo-protein is responsible and may cause anaphylactoid or thrombocytopenic purpura, in others hypersplenism is present and a few follow infiltration or depression of the marrow. When panhæmocytopenia is found marrow involvement is probable, usually following a miliary spread from necrotic

glands or abdominal viscera. In such cases, if there is reason to suspect that the spleen is the major seat of disease, splenectomy should be seriously considered together with streptomycin and P.A.S.

Summary

A case of tuberculosis and thrombocytopenic purpura is described, and the literature relating to the association of the two diseases is reviewed.

The subject is discussed in relation to the forms of tuberculosis which may cause purpura, the ways in which it may do so and the prognosis in such cases; certain conclusions are reached.

We are indebted to Dr. J. V. Dacie of the Department of Hæmotology, Post-Graduate Medical School of London, for helpful suggestions.

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DIAPHRAGMATIC HERNIATION OF THE KIDNEY

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In recent years a great deal has been written on the subject of diaphragmatic hernia. The purpose of the present communication is to describe a case in which diaphragmatic herniation of the right kidney had taken place. Only three similar cases could be found in a study of the literature, thus indicating the rarity of the condition.

Although rare, the condition is capable of accurate diagnosis without operation, provided its existence is remembered; in the present case the patient was subjected to an exploratory operation, which could well have been avoided had excretion urography been carried out during the period of investigation.

Case Report

A. K., male, æt. 52, a coalface worker. This man had a chest X-ray taken in April 1950 because he was complaining of shortness of breath. At this examination a semilunar opacity was noted in the region of the posterior part of the right diaphragm. A diagnostic pneumoperitoneum and pneumothorax were carried out elsewhere, and it was considered at this time that the shadow seen represented a herniation of the liver. The patient's symptoms persisted and he presented himself for re-examination in December 1951. X-ray showed the shadow at the right base to be apparently larger—screening yielded no useful information. Re-examination of the earlier films caused doubt to be thrown on the original diagnosis and exploration was decided upon.

At thoracotomy a typical triangular defect in the position of the pleuro-peritoneal hiatus (foramen of Bochdalek) was found in the diaphragm. The defect was completely covered with diaphragmatic pleura, which formed the hernial sac. Through this sac could be seen bulging the retroperitoneal and perirenal fat. The pleural sac was opened, and the diagnosis confirmed by palpating the right kidney; the entire contents of the hernia were therefore retroperitoneal structures. An attempt was made to close the diaphragmatic defect; this was only partially successful, a matter which caused little concern in view of the innocuous nature of the condition found. The lung was then re-expanded and the chest closed without drainage. Post-operative recovery was uneventful.

Discussion

The first mention of this condition in the literature is by Williams and Tillinghast (1949), who described a case of herniation of the upper pole of the left kidney. The diagnosis was confirmed by operation, when a traumatic hernia of the diaphragm was found containing the kidney. The patient had had a car accident six years previously. Pre-operative pyelography demonstrated a high left kidney, but the provisional diagnosis was neurofibroma.

Bugden (1950) records two cases, in the second of which the correct diagnosis was made pre-operatively. In his first case the right kidney was found to have herniated through the posterior part of the diaphragm. This

patient had had an injury to the right lower chest six months previously. The etiology of this hernia was not described in this paper, but Bugden now considers it to have been a hernia through the Foramen of Bochdalek (Bugden, 1952). In Bugden's second case the hernia was on the left side. There was no history of trauma, and, indeed, the condition was first discovered at routine radiography. Intravenous pyelography demonstrated that the mass seen on X-ray was the left kidney. The hernia was repaired, and the author remarks that the stomach and spleen were easily visible through the peritoneal reflexion, which was all that prevented them from herniating into the chest. For this reason he advocates operation on such hernias, should they occur on the left, commenting that on the right side the condition is probably quite innocuous owing to the protective situation of the liver.

Summary

1. A case of herniation of the right kidney through the pleuro-peritoneal hiatus is described.

2. The literature is reviewed—only three similar cases have been reported.

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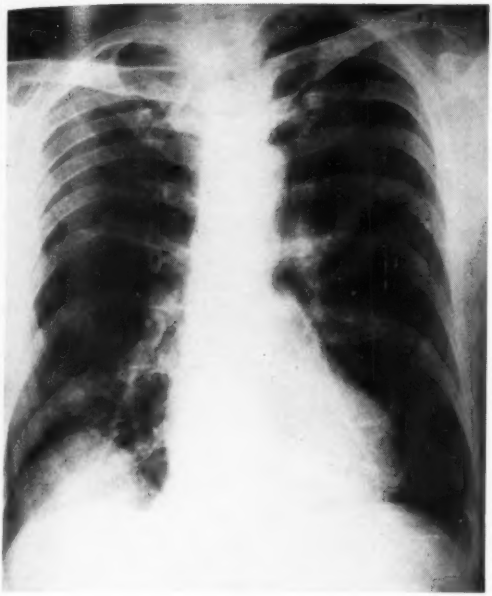

FIGS. 1 and 1A.—Plain radiograph showing posteriorly situated opacity at the right base.

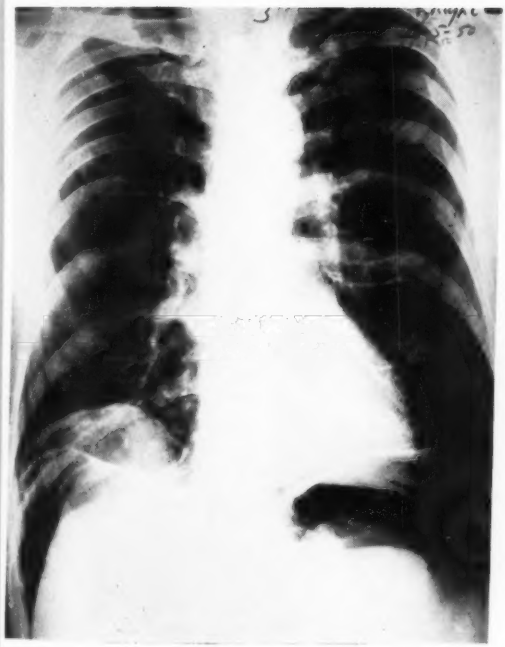

FIG. 2.—After induction of diagnostic pneumoperitoneum.

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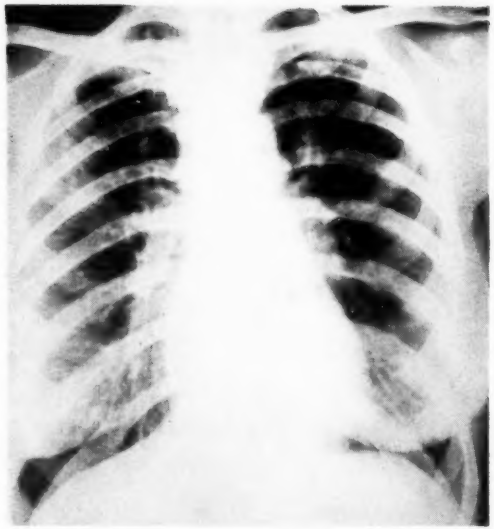

FIG. 1.—Bilateral nodular infiltration with enlargement of the mediastinal glands.

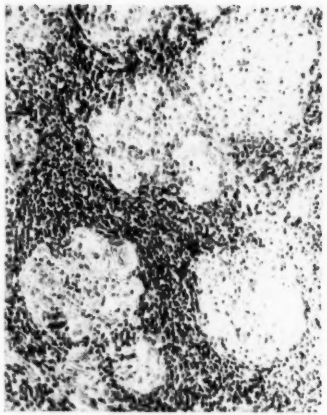

FIG. 2.—Photomicrograph of a portion of the excised lymph node showing the typical sarcoid reaction, with foci of epithelioid cells and a few sparse giant cells. There is no evidence of any caseation.

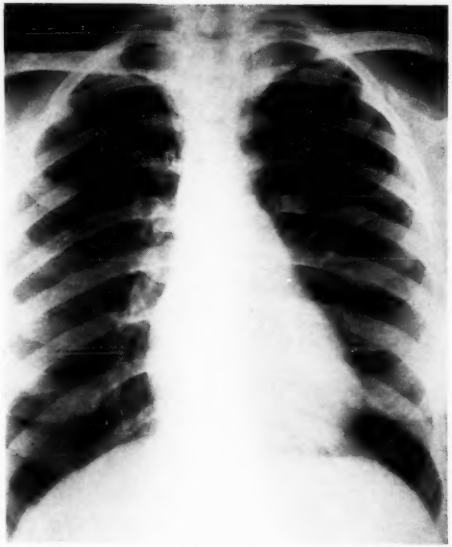

FIG. 3.—The lung fields are clear and the glandular swellings have disappeared.

PROGNOSIS OF SARCOID CHANGES ASSOCIATED WITH ERYTHEMA NODOSUM

By P. O. Leggatt

From Aintree Hospital, Liverpool

Extensive clinical and pathological investigation has done much to clarify the condition known as sarcoidosis, although many questions remain unanswered. One of the major difficulties in this condition, apart from the etiology and treatment, is to find some means of deciding the prognosis in the individual case. It is important, as the majority of these cases are prolonged and may end with a permanent respiratory crippledom owing to progressive diminution of functioning lung tissue, and in a few cases overt pulmonary tuberculosis develops.

Kerley (1942, 1943) described a series of cases of erythema nodosum with hilar adenopathy and extensive but transient pulmonary infiltration. In some of these cases the tuberculin sensitivity was slight or absent. Scadding (1950) described a similar case in a woman aged 51 who had, in addition, cystic changes in the phalanges. Löfgren (1946) reviewed 178 cases of erythema nodosum and in 6 of them there was bilateral hilar adenopathy associated with a miliary pulmonary infiltration. In two of his cases a biopsy was carried out, in one on a lymph gland and in the other a portion of skin was taken. In both the biopsy revealed changes associated with sarcoid.

The following case is thought to be of interest in view of the rapid resolution that occurred.

Case History

F. M., aged 26 years, married. Six months after the birth of her baby she developed on her legs the typical lesions of erythema nodosum, associated with pitting œdema of the ankles and a moderate degree of splenic enlargement. There was no evidence of any abnormality in the chest on physical examination and she did not look or feel ill. Radiologically there was extensive flocculent and nodular shadowing throughout both lung fields, especially in the upper zones, accompanied by some enlargement of the hilar and paratracheal glands on both sides (Fig. 1).

The blood picture and the E.S.R. were both normal. The sputum was negative on guinea-pig inoculation. There were no bony changes in the hands. The tuberculin jelly test carried out nine weeks after the onset of the illness was strongly positive.

Eight weeks after the onset of the illness an enlarged gland appeared on the right side of the neck. This was excised and on examination revealed the typical changes associated with sarcoidosis. There was a marked epithelioid reaction of multifocal origin, the epithelioid cells tended to be arranged concentrically, there were a moderate number of giant cells of the foreign body type, and there was no evidence of caseation (Fig. 2).

It was unfortunately not possible to carry out investigations on the plasma proteins until six months after the onset of the illness, and at that time they

showed no evidence of any abnormality either in their total amount or in regard to the albumin globulin ratio.

The erythema nodosum and the splenomegaly cleared up rapidly and *pari passu* there was a very marked resolution of the radiological picture, including subsidence of the hilar and paratracheal adenopathy. Within sixteen weeks of the original film the radiological picture appeared normal (Fig. 3).

A further film taken two months later also appeared normal. The patient remains in good health and is able to carry out her household duties without difficulty.

Discussion

It is important to decide in the first place whether or not a diagnosis of sarcoidosis has been established in this case. Macleod (1952) is of the opinion that three postulates have to be satisfied before such a diagnosis may be made: there must be a typical radiological picture in the lungs and mediastinum, the patient should not be severely ill, and a biopsy showing evidence of sarcoid tissue is required to complete the picture. Scadding (1950) is of the opinion that the common feature of all sarcoid syndromes is a common histological picture. Although owing to a peculiar state of anergy, a negative tuberculin reaction is not infrequent in cases of sarcoid, many authors will allow cases with a positive tuberculin reaction to be included under the heading of sarcoid. Three of the cases cited by Scadding (1950) were positive to 0·1 mg.-1 mg. of Old Tuberculin. Changes in the bones and the plasma proteins are not at all constant. It is therefore highly likely that my case is one of sarcoidosis, although it may be argued against this that if other glands had been available for excision, or if a liver biopsy had been carried out, caseating tubercles might have been revealed.

My own feeling is that had this been a normal tuberculous reaction it is unlikely that the extensive lung lesions would have cleared up so quickly and so completely. King (1941) in following up thirty-seven cases of sarcoid found that the intrathoracic lesions cleared completely in twenty-three of his cases in periods varying from seven weeks up to three years.

In a simple tuberculous infection of the lungs the hilar adenopathy is most frequently unilateral while in cases of sarcoid it tends to be bilateral: Skiöld (1945), Löfgren (1946). It is also recognised that in cases of sarcoid the pulmonary infiltration is more commonly bilateral.

Many authors suggest that erythema nodosum in association with sarcoid is more commonly found in women of childbearing age (Macleod 1952).

The sarcoid reaction can be produced by a variety of agents, including *Brucella abortus*, Beryllium, and *Mycobacterium tuberculosis*. I believe the pronounced allergy to tuberculin in this case points strongly to the tubercle bacillus as the etiological factor, although it would have been more convincing if she had been known definitely to be tuberculin negative before the onset of her illness.

It is of interest that in a condition with such a variable clinical course some indication of the prognosis in any particular group of these cases is obtainable. It would appear that those cases which present with a sarcoid picture and erythema nodosum have a good prognosis where we may confidently expect the lesions to clear up fairly rapidly without leaving residual damage.

Summary

A case of erythema nodosum with a sarcoid reaction is described. The literature is reviewed in regard to this particular group of the sarcoid syndrome.

It is agreed that the most important features in the diagnosis of sarcoidosis are the radiological changes in the lungs and mediastinal glands, the histology, and the clinical condition of the patient.

It would appear that erythema nodosum in association with a sarcoid reaction carries a very good prognosis.

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IDIOPATHIC PULMONARY HÆMOSIDEROSIS

By J. P. Corridan, P. F. Fitzpatrick and M. Curtin
From the Chest Clinic, City Hall, Cork

Pulmonary changes associated with the deposition of organic iron in the lungs have been described in the following conditions: (i) pulmonary hæmosiderosis, which occurs in adults with mitral stenosis; (ii) idiopathic pulmonary hæmosiderosis, occurring in children and adolescents with hypochromic anæmia.

Cases of pulmonary hæmosiderosis must be differentiated from those showing lung changes due to the inhalation of inorganic iron (siderosis), and the disease should not be confused with hæmochromatosis.

Several cases of pulmonary hæmosiderosis in adults with mitral stenosis have been described (Ellman and Gee, 1951; Lendrum *et al.*, 1950). Idiopathic pulmonary hæmosiderosis was first described by Ceelin (1931). Wyllie *et al.* (1948) described seven cases, five of whom were still alive, and reviewed the clinical and autopsy findings in the seventeen previously reported cases. Further cases have since been reported by Nancekievill (1949), McLetchie and Colpitts (1949), Luther (1949) and Walton and Williams (1951). As the condition appears to have been rarely diagnosed ante mortem the following case report may be of interest.

Case Report

The patient, a girl aged 14 years, was first seen by us in February 1952. She had suffered from infantile eczema up to the age of 3 years and contracted pertussis at $5\frac{1}{2}$ years. Shortly after her fifth birthday she began to complain of increasing lassitude and anorexia. These symptoms continued with remissions until the end of her sixth year, when she became gravely ill and was admitted to hospital. Hospital records show that at that time there was a marked hypochromic anæmia (hæmoglobin 44 per cent.) with cardiac dilatation and pulmonary congestion. She was treated with iron preparations and responded favourably.

In her ninth year she was again admitted to hospital with profound lassitude. Examination at this time showed slight hepatic enlargement, moderate splenomegaly and marked cyanosis. Following her discharge from hospital lassitude persisted with occasional exacerbations necessitating bed rest. She also suffered from dyspnœa and recurrent abdominal pain.

When seen by us in February 1952 she complained of cough, repeated small hæmoptyses, lassitude and dyspnœa.

On examination: The patient, a slightly built, intelligent girl, had a lilac-blue cyanosis of central origin. T. 100° F., P. 110, R. 30, weight 76 lb., height 5 ft. $1\frac{1}{2}$ in. There was an inconstant localised soft apical systolic bruit which disappeared on assuming an erect posture. B.P. $\frac{100}{65}$. Her spleen was palpable 2 inches below the costal margin.

Investigation: X-ray of chest—there was a diffuse dustiness apparent in both lung fields, more marked in the mid-zones and apices. On closer exami-

nation this appearance was seen to be due to countless minute opacities which, in the main, assumed a close pattern that gave rise in places to a pumice-stone appearance. The cardiac outline was normal. Roentgenological bone survey was normal. Tuberculin test was negative (0·005 mg. P.P.D.).

Blood examination:

R.B.C. 3,120,000 per c.mm.
M.C.D. 7·0 μ.
Hæmoglobin 58%. C.I. 0·9.
W.B.C. 9,400.
Lymphocytes 28%, monocytes 3%, polymorphs 67%, eosinophils 2%, basophils 0%.
Reticulocytes 8·5%.
Platelets 245,000 per c.mm.
There was some anisocytosis and slight polychromatophilia.
Bleeding time—3 minutes (normal for method used).
Clotting time—4½ minutes (normal for method used).
Red cell fragility—hæmolysis commenced in 0·44% saline.
Blood group, O, Rhesus positive.
Cold agglutinins, with own cells, present in a titre of 1 in 128.
Blood culture, negative.
Widal, negative.
Wassermann and Kahn reactions, negative.
Spectroscopic examination for methæmoglobin and sulphæmoglobin, negative.
Serum protein 6·5 G. per 100 c.c.
Serum albumin 4·0 G. per 100 c.c.
Serum globulin 2·5 G. per 100 c.c.
Blood cholesterol 145 mg. per 100 c.c.
Icterus index, 5.

E.C.G. normal.
Vital capacity, reduced.
Urine, slight increase in urobilinogen.
Sputum: Blood-stained, non-purulent. Phagocytes containing red cells and hæmosiderin pigment were present in large numbers. Organisms were scanty and consisted of pneumococci and *N. pharyngis*. No acid-fast bacilli or yeasts were seen.

Discussion

In life, as the feature which draws attention to this condition is the unusual X-ray appearance of the lung fields, other conditions giving rise to a similar picture must be borne in mind. This entails a consideration of mitral stenosis, miliary tuberculosis, sarcoidosis, carcinomatosis, Gaucher's disease, streptococcal bronchiolitis, diseases caused by the inhalation of industrial dust and yeast infections.

On reviewing the cases of idiopathic pulmonary hæmosiderosis already described, in addition to the characteristic X-ray changes and hypochromic anæmia, all or some of the following findings were present—hæmoptysis, hepatomegaly, splenomegaly, reticulocytosis, increased urobilinogen, raised titre to cold agglutinins and hæmosiderin in the sputum. The history obtained in the majority of cases was that of repeated small hæmoptyses, recurrent attacks of cyanosis and dyspnœa accompanied by lassitude and infrequent abdominal pain.

The prognosis appears to be unfavourable, as the majority of cases already described have terminated fatally.

The etiology of this condition is still obscure, but Wyllie *et al.* suggest that the primary defect may be in the pulmonary interstitial tissue with capillary stasis, hæmorrhage by diapedesis and deposition of hæmosiderin.

Idiopathic pulmonary hæmosiderosis must still be regarded as a clinical rarity. The case is presented because of this and also because the symptoms have been present over a period of nine years, which seems worthy of record in view of the generally unfavourable prognosis in this condition. It is also of interest that during periods of remission the child has been sufficiently well to attend school although her general health has always been regarded as subnormal.

Our grateful thanks are due to Professor J. M. O'Donovan for the girl's early clinical records, to Professor W. J. O'Donovan for photographic reproductions, and to Dr. J. M. Magner for the pathological examinations and much helpful advice. The case is presented with the permission of Dr. J. C. Saunders, City Medical Officer, Cork.

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PLATE XXIX

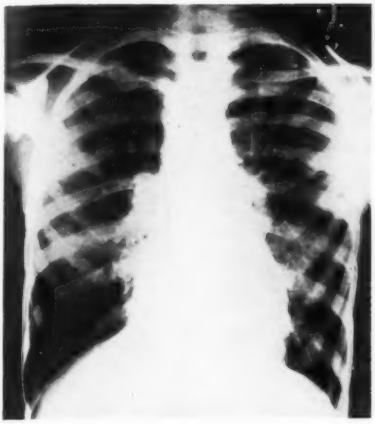

FIG. 1.—X-ray of chest from case of pulmonary hæmosiderosis.

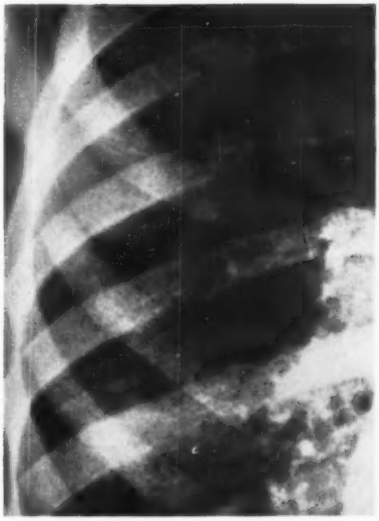

FIG. 2.—Enlarged view of right mid-zone to show up the punctate miliary mottling.

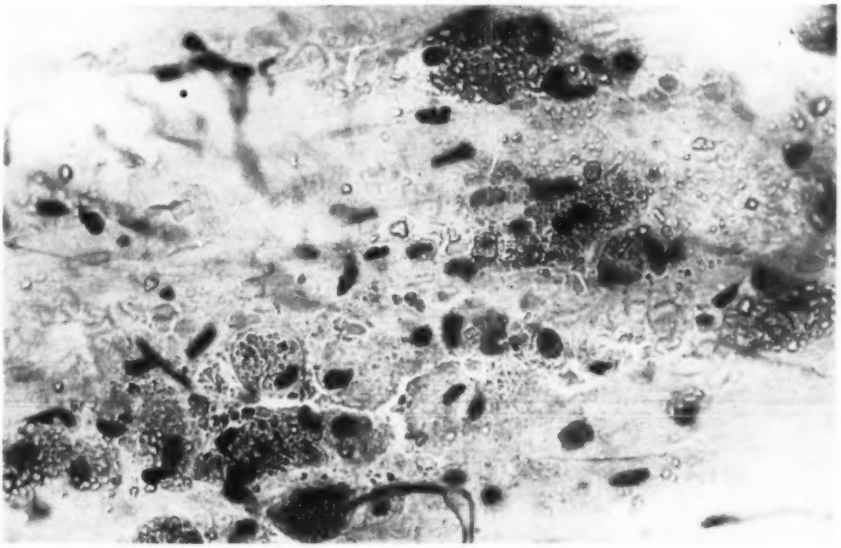

FIG. 3.—Microphotograph of the sputum showing deposits of hæmosiderin.

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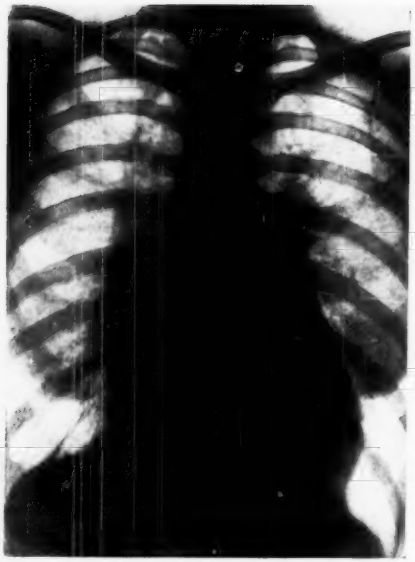

FIG. 1.—X-ray photograph of case of mitral stenosis with pulmonary hæmosiderosis.

FIG. 2.—Detail of lung fields of Fig. 1.

FIG. 3.—Photomicrograph of hæmosiderotic nodule with proliferative arterial changes.

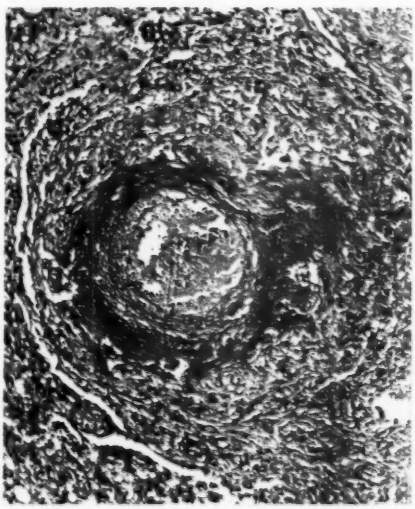

FIG. 4.—Photomicrograph showing necrotising arteriolitis (medial fibrinoid necrosis) with partial obliteration of lumen by organising thrombus.

PULMONARY HÆMOSIDEROSIS IN MITRAL STENOSIS

REPORT OF A CASE WITH PULMONARY ARTERIOLAR NECROSIS

By David Weitzman and O. A. N. Husain

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The advent in recent years of possible surgical relief for stenosis of the mitral valve has greatly stimulated interest in this condition. Attention has also been focused on the associated changes in the lungs. Recently a number of papers, to which reference is later made, have dealt with the problem of pulmonary hæmosiderosis in this disease. We present here a report on such a case, in which post-mortem examination showed, in addition, necrotising arteriolitis restricted to the pulmonary circuit.

Case Report

The patient, a housewife, aged 31, first came under our care in April 1949, with a right lower lobe pneumonia. Mitral stenosis had been diagnosed seven years previously. On admission, auricular fibrillation was also present; this reverted to sinus rhythm as the infection subsided. X-ray examination confirmed right lower lobe consolidation; the cardiac silhouette showed left auricular and pulmonary artery enlargement. There was cardiac failure and associated hepatic enlargement. She responded slowly to treatment with chemotherapy and digitalis.

One year later, miliary nodulation was apparent in all lung fields (Figs. 1 and 2). Fibrillation had become established; signs of mitral reflux, as well as stenosis, were now present.

She remained in fair health and was able to do her housework and shopping until September 1951. She was then readmitted with a further respiratory infection and congestive failure, and died within a few days. At no time during the period of observation was there any hæmoptysis, but occasional examination of the sputum had shown phagocytes containing hæmosiderin.

Pathology

At post-mortem, there was a trace of ankle œdema.

The lungs were firm and distended with air. On section the parenchyma of both upper and lower lobes posteriorly was consolidated and displayed brown induration which exuded a brownish turbid fluid. There were bilateral clear hydrothoraces each of about one litre.

The heart (372 gm.) displayed stenosis of the mitral valve, which admitted one and a half fingers, and left auricular enlargement. There was moderate right ventricular hypertrophy with dilatation of the tricuspid valve and the pulmonary conus. *The liver* was congested; no other viscus was affected macroscopically.

Histology.—Pulmonary hæmosiderosis was confirmed, with scattered nodular collections of siderophages situated mainly in the mid-zones and hilar regions. In these areas the intervening lung tissue often appeared normal.

In the solid œdematous areas posteriorly the pulmonary arterioles and some of the smaller arteries showed a marked fibrinoid necrosis of the media and an infiltration of all coats by inflammatory cells, mostly polymorphonuclears, with a few eosinophils. Still others showed medial hyperplasia and chronic proliferative changes. A few were completely occluded by fibrosis or thrombosis (Figs. 3 and 4).

The parenchyma between these lesions was often grossly œdematous and congested with fibrinous intra-alveolar casts. Myoelastic hyperplasia and early fibrosis of the alveolar walls were also present.

The heart showed characteristic burnt-out chronic rheumatic lesions. The kidneys showed patchy glomerular sclerosis. Other organs showed no significant changes; in particular there was no evidence of any systemic arterial pathology.

Discussion

Although pulmonary hæmosiderosis in mitral stenotics has been known for over twenty years (Wierig, 1927, and Rosenhagen 1928—quoted by the *Lancet*, 1952), the first systematic study of the condition was made by Lendrum, Scott and Park (1950). They also collected the literature for the first time and showed the high incidence of this condition in mitral cases. Ellman and Gee (1951) added further cases, considered the differential diagnosis and set out the various theories concerning its causation. A further review of the subject has recently been made in the editorial columns of the *Lancet* (1952).

In general, the evidence suggests that the condition results from multiple intra-pulmonary hæmorrhages. Lendrum's (1950) work suggested that these arose from varicose broncho-pulmonary capillary anastomoses with rupture into terminal bronchioles; but doubt has subsequently been cast on this by Cudkowicz (1952) and the experimental work of Magarey (1951). Two fundamental questions arise.

Firstly, if hæmosiderosis results from hæmorrhages from engorged pulmonary vessels, why should it occur only in some cases of mitral stenosis and not in others?

Secondly, why are the products of these hæmorrhages not removed? Once present, the condition becomes permanent.

As regards the first question, the increased interest in and awareness of the condition has raised doubts as to whether it really is as uncommon as previously thought. Siderophores ("heart failure cells") are always present in chronic pulmonary congestion (*Lancet*, 1952). These cells are found collected in aggregations around focal hæmorrhages wherever these occur. Similar results are being found in the study of biopsies taken from the lingula of the left lower lobe during operations for mitral valvotomy. Siderophages are seen in practically all such specimens, and are often aggregated around focal hæmorrhages (Enticknap, 1952). Furthermore, Lendrum (1950) drew attention to the fact that radiological recognition during life depended upon the number and distribution of the foci. He stated that the radiological "nodule" was made up of the superimposed shadows of a number of siderotic foci, each individually too small to be apparent on a skiagram. Probably, therefore, not only the spatial distribution, but the technical quality of the radiograph and the thickness of the chest wall may also be important factors.

Finally, awareness of the condition inevitably leads to increasing recognition of the less florid examples.

The second problem, failure of resolution, is still under examination. Pulmonary œdema is considered by a number of observers to play some part. Kerley (1951) thinks that hæmosiderin is deposited because it is insoluble in the alkaline medium of the œdematous lung; and the experimental work of Strassman (1944) showed that induced pulmonary œdema delayed the removal of red cells injected into rabbits' alveoli. However, large pulmonary infarcts are also common in patients with mitral stenosis, and seem to be adequately resolved even in cases with pulmonary œdema. Ellman and Gee (*loc. cit.*) suggest that the question may simply be one of speed, deposits being formed more quickly than they can be removed. The formation of a capsule of fibrous tissue, as shown by Lendrum, would prevent resolution by obstructing the lymphatic drainage. In this connection, the *Lancet* review quoted above points out that iron derived from hæmolysis appears to be mildly fibrogenic in the lung.

It seems equally pertinent to raise the problem of effect as well as that of causation. There does not seem to be any evidence that hæmosiderosis adversely affects the clinical course of mitral stenosis. It is probably less obvious and less widespread in mild cases; but this would merely mean that such cases, with their lesser degree of pulmonary engorgement, are less prone to vascular rupture and focal hæmorrhage. Baker and his colleagues, in their review of surgically treated cases, emphatically state that "it is concluded . . . that hæmosiderosis should not influence the decision to operate one way or the other" (Baker *et al.*, 1952). Ellman in a personal communication reports that two of his cases of mitral stenosis with pulmonary hæmosiderosis have successfully undergone valvotomy, thus confirming this view.

The case described in this paper does not shed any further light on these various problems, but presents one unusual feature in the post-mortem finding of pulmonary arteriolar necrosis. This is present in more than one lobe and often (but not invariably) in association with the hæmosiderosis, each occurring separately in some areas. The arterial lesions vary from a chronic hyperplastic arteriolar sclerosis to a marked acute fibrinoid medial necrosis. Some vessels show considerable polymorphonuclear infiltration (as in polyarteritis nodosa) and occasional complete thrombotic occlusion is present. This last change is, of course, common in pulmonary hypertension.

Pulmonary arteriolar necrosis in mitral stenosis was described by Parker and Weiss in 1936. There is a notable resemblance to the lesions seen in the systemic arterioles in malignant hypertension (Keyes and Goldblatt, 1937; Wilson and Byrom, 1939; Byrom and Dodson, 1948). Symmers (1952) considers very similar lesions to be due to pulmonary hypertension and Lendrum (personal communication) thinks this to be highly probable in our patient. Unfortunately the pulmonary artery pressures were not measured in this case; but the pulmonary artery dilatation seen on the skiagram and the terminal picture of right-sided failure both suggest strongly that pulmonary hypertension was in fact present. A more detailed study of these lesions in this and other cases will be made the subject of a further publication.

Summary

An example of pulmonary hæmosiderosis, in a patient with mitral stenosis, is described. The post-mortem findings include a necrotising pulmonary arteriopathy.

Some of the theories relating to the pathogenesis of hæmosiderosis are reviewed and discussed. Attention is drawn to the frequency of the condition.

ACKNOWLEDGMENTS

We take this opportunity of expressing our gratitude to Dr. Philip Harvey for permission to publish the data in this case, and to Dr. Philip Ellman for his interest and encouragement. We are much indebted to Professor Lendrum for a great deal of helpful advice and criticism. Our thanks are also due to Drs. Signy and Gillespie for assistance with the pathology and to Dr. Enticknap for allowing us to include unpublished material.

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SEGMENTAL ATELECTASIS

By F. E. Saxby Willis
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Pulmonary collapse conforms to two main types. It may be massive and affect a large portion of or an entire lung. Such a condition most often follows abdominal operations and constitutes the clinical picture of post-operative pneumonia. Other causes of massive collapse are trauma, as in gunshot wounds of the chest wall, paralysis of the muscles of respiration, as in diphtheria and bulbar forms of poliomyelitis. Blockage of a main bronchus may be caused by a foreign body or a bronchogenic carcinoma and lead to massive collapse of a lobe, or it may be caused by increased intrapleural pressure, as in spontaneous pneumothorax and large pleural effusion. Weakness of the muscles of respiration, as in conditions of extreme debility and in patients confined to bed for long periods, may lead to basal atelectasis.

Another type of this condition is that affecting smaller areas or segments of a lung, often complicating bronchitis or bronchopneumonia. Segmental atelectasis may follow pneumonia or other inflammatory conditions. One specific cause to which considerable attention has been directed is "epituberculosis," which may be the result of a non-caseating tuberculous pneumonia or may be due to an area of collapse secondary to bronchial obstruction caused by enlarged glands.

If the areas of segmental collapse are inflammatory they are dark in colour owing to vascular congestion, have a definite margin and are firm to pressure. On section they are found to be airless and sink in water.

The symptomatology is dominated by the associated or causative condition. Usually there is fever at the onset where the atelectasis is infective in origin, but in cases where the cause is purely obstructive there may be little or no constitutional disturbance.

In cases where large areas of lung are collapsed there will be marked dyspnœa, usually relieved by rest in bed. The respiratory and pulse rates are increased and there may be considerable respiratory distress. Cyanosis is unusual, cough is slight, and there is no sputum.

Physical signs vary according to the area of lung involved, being indefinite or absent in small areas and manifesting dullness to percussion and absent breath sounds only where larger areas are collapsed.

Diagnosis is established by radiographs which should include postero-anterior, lateral and in some cases antero-posterior lordotic views. These will show loss of translucency corresponding to the area of segmental collapse (more usually triangular in outline) and displacement of the interlobar fissures towards the lesion.

In a series of 130 out-patients at the Royal Chest Hospital early in 1951, 15 were diagnosed as cases of acute non-tuberculous pulmonary inflammation with or without accompanying atelectasis. The patients attended as walking cases, all but 6 being afebrile. Investigation consisted of clinical, X-ray and

pathological examination, including bacteriological examination of sputum (three for tubercle bacilli).

In no case was rigor noted at the onset. In 5 cases there had been excessive sweating.

Although cough was noted in all cases, sputum was purulent in only 5 and in the remainder was either absent or scanty and mucoid. Chest pain was noted in 8 cases and was shown by X-ray and physical signs to be due to pleurisy. In no case were physical signs typical of pneumonic consolidation present. In 2 cases signs of generalised bronchitis were present. A pleural rub was heard in 3 cases. In only 1 was the patient ill enough to need admission to hospital.

The presence and extent of the pulmonary lesion was revealed by routine X-ray examination which showed, particularly in the lateral views, localised consolidation and, in some cases, associated atelectasis, conforming to the picture of a segmental pneumonia.

The right lung was affected in 8 cases, the left in 5, and in 2 cases segments of both lungs were involved.

Serial radiographs taken at intervals of one or two weeks showed eventual complete resolution, although this was sometimes delayed for several weeks. Cases of inflammatory origin showed thickening of the interlobar septum and peaking of the diaphragm.

Blood examinations were made in most cases, and showed the E.S.R. to be raised in 6 cases only, and this fell to normal within two weeks in all except the case admitted to hospital.

Anæmia was found in 2 cases only, the blood count becoming normal in two weeks.

Differential white cell count showed in 2 cases an excess of lymphocytes and mononuclear cells consistent with virus infection. In 2 other cases there was a relative excess of polymorphonuclear cells suggestive of an acute pyogenic infection.

In 3 cases there was neither fever nor a raised E.S.R. Two of them gave a history of bronchitis. In these cases it is suggested that the X-ray appearances were due to segmental atelectasis caused by blockage of a bronchus or to aspiration pneumonia, in which a plug of infected mucus had caused local inflammatory reaction.

In only 5 cases was a bacterial cause detected and in these there were associated fever and constitutional disturbance.

In the remaining 10 cases the absence of sputum and of constitutional disturbance suggested that the illness was probably due to atelectasis of non-infective origin.

The characteristic radiological appearances of segmental atelectasis have been described in detail by Lubert and Krasse (1951).

They suggest that the lung should be considered in its major subdivisions as semi-rigid rather than purely elastic structures. Except for the alveoli, collapsed pulmonary tissue cannot be regarded as a "deflated balloon." When a lobe collapses a space is created which must be filled by diminution in size of the hemithorax, displacement of mediastinal structures, elevation of the diaphragm, expansion of the adjacent pulmonary tissue or a combina-

tion of all these factors. Collapse alters the shape of the affected lung segment. The usual three-dimensional conical configuration is replaced by a flattened triangle with the apex at the hilum. The broad part of this triangle follows the curve of the parietal pleura with the base (interlobar septum) lying against the lateral chest wall.

The lobes do not merely shrink to become smaller replicas of themselves but change from a three-dimensional pyramidal or conical figure to a two-dimensional triangle and flatten along the mediastinal and parietal pleura. With continuation of the collapsing force, they move along the curved surface of the chest wall toward the hilum, the direction of the movement depending on the natural configuration and position of the lobe and the presence or absence of modifying factors.

In cases of segmental pneumonia, with or without atelectasis, the changes are somewhat different. The appearances of consolidation tend to clear gradually, but sometimes there is considerable delay.

In inflammatory lesions of the lung parenchyma the shadowing is more irregular and mottled. Thickening of the pleura develops and the position of the interlobar septa indicates the degree of associated collapse. Where the lower lobes are affected the associated pleurisy causes peaking of the diaphragm.

Xalabarder (1951) believes that the lung possesses its own mobility and does not follow passively the movements of the thoracic walls. He gives clinical and radiological reasons for regarding atelectasis as a phenomenon of active contraction of the pulmonary parenchyma which need not involve the previous occlusion of the bronchial tree.

Post-operative Pneumonia

A detailed description of post-operative pneumonia is given by Baker, Roeltig and Curtis (1951), who hold that large bronchi are rarely occluded by solid plugs of secretion. The bronchial tree is cleansed by respiratory tract secretion, evacuated by ciliary movement and the expulsive action of the bronchi. When secretion is excessive removal is aided by the respiratory fluid which, altered by pathological changes, produces bronchial occlusion leading to atelectasis. Discussing the secretary properties of the bronchial glands and goblet cells, they conclude that post-operative atelectasis occurs particularly in patients who have a highly viscid bronchial secretion, due to factors such as asthma, upper respiratory infection, chronic bronchitis and bronchiectasis. They conducted an investigation into methods of producing a more physiological bronchial secretion and quote Holinger (1941) who found that in patients with bronchiectasis the mode of action of the common expectorants, including potassium iodide, ammonium chloride, emetine chloride and steam, was to lower the viscosity of the bronchial secretion.

To prevent the development of post-operative atelectasis sodium iodide 1 gramme b.d., particularly when given intravenously, was found to be more effective than the routine pot. iod. orally.

Ariel (1941) working with radio active iodine found that the lungs and thyroid gland collected most of the radio-active iodine injected. Tuft and Levin (1942) demonstrated that iodides given either orally or intravenously are selectively excreted into the bronchi within 15 to 20 minutes.

In some cases iodides alone are not sufficient, and bronchoscopic or tracheo-bronchial aspiration with a catheter may be needed when obstructive atelectasis has developed.

Aspiration Pneumonia

Robertson and Forgan Morle (1951) have reported a series of 500 cases of atypical pneumonia confined to local regions of the lung, which, they suggest, are due to aspiration pneumonia. The acceptance of an aspiration basis for "primary atypical pneumonia" provides an explanation for its obscure epidemiology, and its infectivity and method of spread are like those of an upper respiratory tract infection. This may also account for some cases of segmental atelectasis of which an example from my own series may be quoted, that of a boy of five with huge infected tonsils and adenoids who was admitted to hospital for tonsillectomy. As he had a troublesome cough and slight fever his chest was radiographed and showed bilateral collapse of both lower lobes. Bronchoscopy showed both lower main bronchi to be filled with cheesy pus completely obstructing the lower bronchi. The condition was complicated by a bilateral antral infection. The X-ray appearances in this case might well have been mistaken for pneumonia and were indeed originally reported on as such, but a bronchogram demonstrated the obstructive origin of what was actually atelectasis.

Congenital Atelectasis

This is found sometimes in new-born infants but is more often overlooked until later in life when it may be found to co-exist with such conditions as cystic disease of lung (Saxby Willis and Almeyda, 1943). The condition of congenital agenesis may give rise to a similar clinical picture although usually in this there is more asymmetry of the chest (Brimblecombe, 1951). Diagnosis is established by the persistence of the abnormal physical signs and the radiological picture.

Post-pneumonic collapse of the right lower lobe relieved by bronchoscopic aspiration is described by Miller (1950).

He suggests that bronchoscopic examination is indicated as a routine measure in all cases of segmental pneumonia or atelectasis which have not resolved or shown signs of re-expansion.

The right middle lobe is stated by Rubin and Rubin (1950) to be particularly liable to atelectasis. They describe what they term the shrunken right middle lobe or "middle lobe syndrome," illustrating from 16 cases of this condition, not including cases caused by neoplasm, pneumonia, aspiration of foreign body or tuberculosis where the condition is incidental to a more widespread process.

In 6 cases the right middle lobe was shrunken and bronchiectatic. In one case the bronchiectasis was only discovered at autopsy.

In 5 cases bronchoscopy revealed a shrunken lobe associated with tuberculous bronchitis. In 2 of these encapsulated serous fluid was present between the middle and lower lobes which, on aspiration, revealed acid-fast organisms.

One patient expectorated what he thought was a broken tooth but was found to be a piece of broken lymph node. Two years later acid-fast organisms

were found in the sputum. Another patient coughed up a broncholith while being prepared for a middle lobe lobectomy.

They conclude that a pre-existing tuberculous focus, probably in the regional lymph nodes, may lead to involvement of the right middle lobe. Middle lobe shrinkage may, however, occur with or without bronchiectasis as the result of pressure exerted on bronchi by cicatrising non-tuberculous lymph nodes as well as from other causes.

They point out the importance of bronchoscopy in the differential diagnosis of a shrunken right middle lobe, particularly since the majority of cases in their series were of the cancer age and it is important to exclude bronchogenic carcinoma.

They stress the frequency with which tuberculous bronchitis is encountered in right middle lobe disease.

Bronchography demonstrates the presence of bronchiectasis or bronchial obstruction beyond bronchoscopic view. Four cases showed variable degrees of cylindrical or saccular bronchiectasis and in one there was involvement of the lingula as well as of the right middle lobe bronchus.

They conclude that the right middle lobe is more subject to compression by enlarged or cicatrising lymph nodes, and quote Brock (1950) in this connection. They describe how the right middle bronchus, as it leaves the main stem, forms an acute angle. At the point of bifurcation the bronchus is surrounded by lymph nodes which drain both the middle and the lower lobes. Should the bronchial channel become obstructed the lobe collapses, leading in many cases to bronchiectasis.

Coulter (1950) has demonstrated that occlusion of the air passage supplying a unit of the lung which does not also communicate with another air passage by collateral respiration will result in atelectasis by absorption of the trapped air.

Experiments were carried out to determine whether a valvular obstruction would produce atelectasis. It was found that a one-way valve placed in the bronchus produced rapid collapse of the corresponding portion of the lung.

He believes that practically all cases of atelectasis can be explained on the basis of bronchial obstruction.

Among the rarer causes of bronchial obstruction leading to atelectasis may be mentioned innocent growths, particularly adenoma and polypus of which I have recently had a case under my care. The history was one of troublesome cough with purulent expectoration for the past nine months, in a man aged 36. Clinical examination showed impaired resonance and diminished air entry with râles at the base of the right lung. Radiological examination showed shadowing in the corresponding area. Bronchographic examination showed failure to fill in the posterior position of the right lower lobe. Bronchoscopy revealed partial obstruction of the lower lobe bronchus caused by a polypus which has since been successfully removed.

Diagnosis

In cases of segmental atelectasis revealed by radiological examination it is important to seek and determine the cause and extent of the lesion. It is important to exclude such causes as foreign bodies and bronchogenic carcinoma.

This entails routine bronchoscopic examination in addition to serial X-ray examination, which in cases of difficulty should include a tomograph and bronchograph. Purely obstructive, in contrast with inflammatory, causes are indicated by normal temperature and E.S.R. and normal leucocyte counts. It is particularly important to exclude tuberculosis as a cause.

Treatment

This will obviously depend on the cause in individual cases, the most common of which is probably post-operative bronchial obstruction by inspissated mucus. To prevent this it is important that the patient should be in the semi-recumbent or sitting-up position as soon as the surgical condition permits. The administration of expectorants is probably of little real value. Intravenous injections of sodium iodide gr. 1 has been referred to above as a means of preventing the development of atelectasis and resultant pneumonia. Neo-adrenaline has been used for the same purpose.

In cases of inflammatory obstruction in the convalescent stage breathing exercises and other physical means directed to the promotion of re-expansion should be tried. If, however, there is, after a week or two, a residual atelectasis, bronchoscopy is indicated.

The possibility of tuberculosis as the causative condition should not be forgotten and in such cases treatment will be on sanatorium lines supplemented by specific measures.

If bronchogenic carcinoma is found to be the cause surgical treatment should be considered and, if it is not suitable, deep X-ray therapy or implantation of Radon seeds into the obstructed bronchus may be indicated.

Summary

An attempt has been made to describe and distinguish the main causes of pulmonary atelectasis, particularly in its segmental form. Radiological examples of the condition resulting from these various causes are appended. The diagnosis and treatment of this condition in its varying manifestations have been briefly discussed.

Illustrations of Cases of Segmental Atelectasis

Figs. 1 and 2 Pneumonia with atelectasis R.M.L.*
Figs. 3 and 4 Pneumonia with atelectasis anterior segment R.U.L. P.A. and lateral views.
Fig. 5 Atelectasis due to epituberculosis R.U.L.
Fig. 6 Atelectasis R.M.L. and L.L.L. due to U.R.T. infection and obstruction by inspissated pus.
Fig. 7 Atelectasis R.U.L. due to bronchogenic carcinoma with lung abscess.
Figs. 8 and 9 Atelectasis of left lung due to gross cystic bronchiectasis with supervening bronchogenic carcinoma.
Fig. 10 Specimen showing atelectasis due to cystic disease of the lung (subsequently developing bronchogenic carcinoma).
Figs. 11 and 12 Atelectasis R.U.L. due to congenital cystic disease. Lobectomy was successfully performed.

*Key. R.M.L. = right middle lobe
L.L.L. = left lower lobe
R.U.L. = right upper lobe
U.R.T. = upper respiratory tract

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REVIEWS OF BOOKS

Tubercle Bacillus and Laboratory Methods in Tuberculosis. By M. A. Soltys in collaboration with C. A. St. Hill and I. Ansell. E. and S. Livingstone Ltd. 1952. Pp. viii+212. Price 20s.

The first 68 pages in this book are devoted to a description of the bacillus. Necessarily therefore the information given is scrappy, and important work may be dismissed in a few lines. However, the bibliography is comprehensive and international, so that the section would be useful as a starting-point in the search for information which the text itself cannot provide.

Laboratory methods are discussed in fuller detail in the second part. There are descriptions of the collection, technical treatment, staining, cultural and animal inoculation methods employed in demonstrating the bacillus in all types of specimens. Now that the isolation of scanty bacilli is of such importance, not only in the diagnosis but in the treatment of tuberculosis, it is perhaps regrettable the authors do not indicate their personal preferences among the many methods given.

As a great deal of time and effort must now be spent in determining the sensitivity of the bacillus to streptomycin it is disappointing to find so much space devoted to the M.R.C. fluid medium method with its well-known fallacy. The use of Herrold's medium, in spite of its technical difficulties, is described at length, when a fuller discussion of the results obtained by the incorporation of graduated quantities of the drug in such a well-tried and convenient medium as Loewenstein and Jensen would have been more useful.

K. F. W. Hinson.

Lung Abscess. By R. C. Brock. Blackwell Scientific Publications, Oxford. February 1952. Pp. 197. 60 figures. 35s.

From time to time it is wise to pause and take stock of the evolution of each branch of surgery. Sporadic articles, new contributions, day-to-day policies modify traditional teaching; practices become established not always well supported by the facts; and a diagnosis that passed well enough in 1942 proves woefully inadequate in 1952. Such a stage has been reached in the treatment of abscesses of the lung, and Mr. R. C. Brock provides in his new monograph a lucid review of the whole subject in the light both of present-day knowledge and of fundamental principles.

The book opens with a brief and clear account of the morbid anatomy, and the absolute necessity for accurate localisation of the affected lung segment is stressed. Pathologically the prime cause of most lung abscesses is bronchial obstruction; and their course thereafter depends upon the form this takes and the nature of the organisms which commonly accompany it. " It cannot be too strongly emphasised nor too often repeated," he says, " that ' lung abscess ' is not an entity." Of 318 cases, not associated with growth, he found about half foetid—*i.e.*, anaerobic; and half non-foetid. Either may be primary, but as a rule is secondary to some determinable cause; and in the aerobic group are to be distinguished the important and common staphylococcal abscesses so often encountered in children, and the less frequent but characteristic Friedländer variety. To both of these and to their differing management he devotes a chapter, while a further section deals with the three types of abscess associated with bronchial carcinoma and which accounted for 18·9 per cent. of the total

392 cases seen from 1942 to 1950. With sufficient care a cause can be identified for over 70 per cent. of abscesses: and all too often in this country ill-judged dentistry, with incompetent anæsthesia, carried out in the indefensible upright dental chair, can be found guilty (7 per cent. of Brock's cases, 11·3 per cent. of Stern's).

Two themes dominate this book: the need for *real* diagnosis, etiological, bacteriological, anatomical, as opposed to the affixing of a slipshod label; and the recognition that lung abscess is always a dangerous disease with a high mortality and a still higher morbidity rate, calling for prompt and definitive surgical relief as soon as it is apparent that conservative measures are inadequate. Both these things needed saying and Brock says them forcibly with a wealth of illustrative and statistical evidence. When analysing the results of " medical " treatment it is essential that those cases coming to surgery in the same period be counted among the failures, and their number added to the total. If this is done it will be found expectancy fails, he says (even with chemotherapy and antibiotics), in some 60-80 per cent. of patients. External drainage may be imperative when acute abscesses contain lung sloughs; but the process has in the past been associated with a mortality of 25 per cent. 272 recently reported primary resections for lung abscess by contrast yielded good results in 82 per cent., with an average mortality of only 9 per cent. " The mind of the thoracic surgeon today is turning away from external drainage to resection of lobe or lung when resolution is incomplete after six to eight weeks. In contrast, the minds of many physicians are just beginning to turn towards external drainage when this stage is reached. Could there be any greater contrast in the approach to a major problem than this ? The one conception is that the proper time for surgery has been reached, the other that the proper time for conservative surgery has already passed."

GEOFFREY FLAVELL.

Causes and Prevention of Tuberculosis. By BRICE R. CLARKE, with a chapter on Mass Miniature Radiography by Joseph Ritchie. Edinburgh: E. and S. Livingstone Ltd. 1952. Pp. viii+288. 32s. 6d.

This very important book surveys a wide field. " Causes " include an account of the natural history of the tuberculous process; an examination of endogenous reactivation and exogenous superinfection; of the influence of race and heredity; food, overcrowding, epidemiology and bovine tuberculosis, etc. " Prevention " includes the prevention of infection; the ascertainment and prevention of disease; immunisation; and treatment and rehabilitation in so far as they affect prevention.

In all this the extraordinary difficulty of disentangling one causal factor from another is done with great skill and with only a minimum of repetition. Where repetition is unavoidable, all necessary cross-references are given.

Each chapter is based on a painstaking analysis of a vast amount of published work (over 700 authors are quoted). This means that whilst Dr. Clarke has accordingly performed an enormous service to the student of tuberculosis, the very method adopted makes the reviewer conscious of three defects. Firstly, Dr. Clarke is so busy giving us the views of others that he modestly omits to give us enough of his own authoritative opinions (a welcome exception is the account of his experience in Belfast in relation to the effects of overcrowding in tuberculosis). Secondly, the method adopted gives him little chance to provide adequate chapter summaries so helpful in scientific work (an exception here is his summary of the value of B.C.G.). Thirdly, the length of the analysis

of each subject tends to be in direct proportion to the number of relevant publications, and this results in a certain lack of balance. For instance, the chapter on food and tuberculosis is unexpectedly the largest in the book, and occupies as much space as the whole of the three chapters on prevention of infection, prevention of disease and immunisation against tuberculosis.

Here and there, as in any book, the reviewer may not agree with the author. Rudbäck (1948), for instance, is given the credit (p. 52) for establishing that tuberculous bacilluria occurred in primary tuberculosis in Swedish children, but this was demonstrated some years earlier by Munro (1941-44) in this country.

Very rarely a misprint creeps in. Koch's name is wrongly spelt (p. 265), as also is Sasano's (p. 215), and the reference to the latter's paper is omitted from the chapter bibliography.

But if there are a few points to criticise, it is quite impossible to list all there is to admire in this work. Such a book has been needed for years. Every tuberculosis specialist will find it essential. The book has come at the right time and clearly from the right source.

CHRISTOPHER CLAYSON.

Disorders of the Heart and Circulation. Edited by ROBERT LEVY, M.D. Baillière, Tindall and Cox. 1951. Pp. xv+944. Price 91s. 6d.

The Editor tells us " this book is planned to cover all that is known about the causation and treatment of heart disease." There are 34 chapters, well printed with excellent illustrations, graphs and tables, and the contributors bear distinguished names in the cardiovascular field of American medicine. Most of the chapters were written for Nelson's Loose-Leaf Medicine, before the Editor-in-Chief, Dr. Walter W. Palmer, died in 1950. Dr. Robert L. Levy, the distinguished New York cardiologist, who had collaborated with Dr. Palmer in assembling the material, has edited this volume of monographs, some revised, with additional chapters, as a tribute to Dr. Palmer. The book is compiled in the spirit of the second half of the twentieth century. The scope of the book, addressed to students and specialists by contributors with authoritative first-hand knowledge, is above elementary level, and diverse aspects are expounded in reasonable balance. The references are adequate for further reading by those interested in one or more special subjects. The following samples may give some indication of what the Editor and other contributors have provided for the reader:

Maude Abbott's original classical description of congenital heart disease has been included in the appropriate section.

The chapter on pulmonary heart disease by Sprague of Harvard University should interest the readers of this Journal. He has given a clear exposition of the hæmodynamics of the lesser circulation in health and disease. Embolism, the source usually phlebo-thrombosis, is stressed as the commonest cause of acute cor pulmonale. The initial symptoms and signs are embolic; at a later stages, those of infarction. The electrocardiographic evidence of pulmonary heart disease is well displayed and its early recognition, so often transient, is emphasised as of great diagnostic value. The treatment recommended is oxygen, morphia and coramine; intravenous papaverine is considered a hazardous risk; venesection is contra-indicated, digitalis is ineffective, embolectomy rarely succeeds, anti-coagulant therapy should always be considered, and femoral ligation if performed must be bilateral.

There is an acceptable etiological classification of chronic cor pulmonale, based on elevation of pulmonary venous pressure, as in the secondary effect of

mitral stenosis; obstruction to pulmonary blood flow from structural changes in the lungs, and by including emphysema the term " emphysema heart " is accepted with some qualification; as a third and much rarer cause primary obliterative vascular sclerosis. Congestive failure may be slow in its development, with a short and rapid terminal course from right ventricular weakness.

Diseases of the pericardium are described by Wood and Krumbhaar of Philadelphia under the following headings: acute pericarditis, pericardial effusion, pyopericardium, chronic cardiac constriction. Bellet's observation that there is no clinical evidence of tuberculosis elsewhere in a third of the cases of tuberculous pericarditis is supported. Primary tuberculous disease of the pericardium is regarded as a rarity, pericardial constriction a natural sequel of this infection, cases of " unknown etiology " may be tuberculous. The prognosis is always grave, but the pathological process can be arrested and recovery does occur. Streptomycin has given a better outlook to treatment and made safer pericardial surgery before infection has become quiescent. The value of chemotherapy and antibiotics in lessening the incidence of acute purulent pericarditis is stressed, and paracentesis and sulphonamides have in some measure replaced incision and drainage.

T. F. COTTON.

The Care of the Ageing and Chronic Sick. By A. P. THOMSON, C. R. LOWE and THOMAS MCKEOWN. Published for the Birmingham Regional Hospital Board, by E. and S. Livingstone, Ltd., Edinburgh and London. 1951. Pp. 133. Price 17s. 6d.

There are about five million people in Great Britain above 65 years of age. Of these, roughly 150,000 are accommodated in institutions of one kind or another. It is estimated that if present trends continue the number of people over 65 in Great Britain a quarter of a century hence will be 8·5 millions and the social problem of old age will be the most difficult of any that threaten Western civilisation.

The personnel of the present communication is evidence of the interest with which the problem is being approached in Birmingham and the ability of those who are undertaking the task. Professor Thomson has all the attributes demanded, as a clinician, a philosopher, an administrator and, one may add, a philanthropist. His major contribution is the reproduction of his Lumleian Lectures for 1949 in which all aspects of the subject are considered.

On the clinical side he emphasises what is only too certain, that the exclusion of the aged and chronic sick from the wards is responsible for serious defects and deficiencies in the training of medical students, although such material constitutes a considerable proportion of the work in general practice. They are regarded by general hospitals as a nuisance, their prolonged stay reducing the admission of subjects of greater clinical interest with more dramatic possibilities and results as well as greater examination value.

Admittedly the speciality of geriatrics has more of a social than a medical application, yet the latter is by no means to be neglected, especially in the direction of rehabilitation.

The most pressing problems of old age are poverty, solitude and infirmity. The contributions by Dr. Lowe and Professor McKeown include a number of statistical tables of considerable interest and value. They range from such obvious details as age and sex incidence to the duration of stay in hospital and the attitude towards leaving hospital, especially in relation to duration of

stay. The outstanding symptom is incontinence and its considerable improvement whan a bed-fast patient is encouraged to get up. Occupation in hospital, frequency of visits and similar details are correlated with the available domestic resources outside. For it is estimated that fully 60 per cent. of the patients did not require hospital treatment at all; they had no need of skilled nursing or of frequent medical attention. They occupied beds because they were homeless and had no relatives or friends able and willing to care for them. Much could be achieved if hospital beds could be spared by provision of alternative accommodation and simple supplementary attention in their own homes. But extensive investigation showed that less than 20 per cent. of the houses visited attained a reasonable standard of home care for an invalid, a bath and an adequate supply of hot water and indoor sanitation. The squalor and misery displayed indicate that the social problem created by the increasing number of chronic sick extends far beyond the difficulty of providing adequate medical and nursing services.

This is a noble enterprise calling for much self-sacrifice, no kudos and little return beyond the satisfaction of achievement. It may be said that a special flair for such work is indispensable, a character to appreciate the ethical advantage (even if a ruthless economy condemns the enterprise), and at the same time an enthusiasm to investigate senescence as a practical feature, to refuse to accept fatalistically a presumably inevitable deterioration, but to investigate to what extent certain of the changes are in fact reversible.

ADOLPHE ABRAHAMS.

Non-Pulmonary Tuberculosis of Bovine Origin in Great Britain. By G. S. WILSON, J. W. S. BLACKLOCK and LILIAN V. REILLY. The National Association for the Prevention of Tuberculosis. Pp. 107. Price 16s.

This book gives an account of the investigations made during the war years 1943-1945, under the direction of the Medical Research Council, into the incidence of human tuberculosis of bovine origin in England, Wales, Scotland and Northern Ireland. Pulmonary tuberculosis was excluded. The co-ordinators of the scheme were Professor G. S. Wilson, Professor J. W. S. Blacklock and Dr. Lilian V. Reilly, and their reports are published in detail. In addition, a summary report dealing with England, Wales and Scotland is given by Professors Wilson and Blacklock. The English, Welsh and Northern Ireland reports appeared originally in the *Journal of Hygiene*. The Scottish report is published here for the first time. No two of the reports, for reasons discussed by the authors, are strictly comparable, but they all contain a wealth of carefully analysed information which is worthy of careful study by all who are interested in the epidemiological and social problems of tuberculosis, and the N.A.P.T. has done a service in producing this publication. All investigations of this kind have been milestones in progress, and it is probable that this book is already a milestone, for the acceleration of the pure milk campaign, the attested herds scheme and the coming into operation of the Milk (Special Designations) Scotland Order, 1949, have led to further improvement in the herds and to greater safeguards against infection of the public. This aspect of tuberculosis is in fact rapidly coming under control in many parts of the country. In the summary report for England, Scotland and Wales (p. 16), already referred to, the authors have confused annual notifications with numbers on the " live " register which are stated correctly in the Scottish report (p. 89), and the statement that during 1943-44 there

was a yearly notification of about 10,000 cases of surgical tuberculosis leads to the further startling statement that we arrive at a figure of between 3,000 and 4,000 as the number of notified cases in Scotland attributable to bovine tuberculosis. This confused paragraph should be rewritten.

CHARLES CAMERON.

BOOKS RECEIVED

The following books have been received and reviews of some of them will appear in subsequent issues.

Australasian Annals of Medicine. Volume 1, No. 1. May, 1952. Journal of the Royal Australasian College of Physicians, 145, Macquarie Street, Sydney.

Medical Biographies—the Ailments of 39 Famous Persons. By Philip Marshall Dale. University of Oklahoma Press. Pp. 259. $4.

NAPT Handbook of Tuberculosis Activities in Great Britain and the Commonwealth. Fourteenth Edition. Pp. xii+370. 30s.

The Protection of the Nurse against Tuberculosis. By F. A. Simmonds. NAPT. Pp. 90. 10s. 6d.

Non-pulmonary Tuberculosis of Bovine Origin in Great Britain. By G. S. Wilson, J. W. S. Blacklock and L. V. Reilly. NAPT. Pp. 108. 16s.

Modern Electrocardiography. Vol. 1. The P-Q-R-S-T-U Complex, by E. Lepeschkin. Pp. xiv+598, with figures. Price 91s. 6d. net.

Help Yourself Get Well. A Guide for T.B. Patients and their Families. By Marjorie McDonald Pyle, M.D. Heinemann. 1952. Pp. xv-143. Price 12s. 6d.

St. Thomas's Hospital Reports. Volume 7, second series, 1951. Pp. 184. 10s. 6d.

The Longitudinal Study of Families as a Method of Research. By Jean Downes. Pp. 18. Milbank Memorial Fund, 40 Wall Street, New York 5.

Papworth's Progress. Report for 1951. Pendragon (Papworth) Press.

Brompton Hospital Reports, Vol. XX, 1951. Research Dept., Brompton Hospital. 12s. 6d.

The Principles and Practice of Medicine. By L. S. P. Davidson. E. and S. Livingstone, Ltd. 1952. Pp. 919. 32s. 6d.

Traité de Médicine. Volume VI, "Tuberculose Pulmonaire." By M. Bariety, A. Dufort, P. Galy, E. Hautefeuille, J. le Melletier, J. Leroux-Robert, J. P. Nico, A. Meyer and A. Ravina. Masson et Cie, Paris. 1952. Pp. 558. Price £4 10s.

REPORTS

MINISTRY OF HEALTH

Tuberculosis in England and Wales

We give below excerpts from a report from the Ministry of Health [M.H. Reference Note 5 (revised July 1952)].

Incidence and Trend

Since the war there have been important developments in the treatment of tuberculosis which bear promise of hastening the day when the disease will be brought under control. Two new drugs, streptomycin and para-amino salicylic acid, are proving powerful weapons against the tubercle bacillus. Better diagnosis, the increasing use of mass radiography to search for unsuspected cases in a community and so reduce the unknown reservoir of infection, the steady addition of beds for T.B. cases, the extension of home treatment both before and after admission to sanatoria, and the improved co-ordination of all the anti-tuberculosis forces, are having a beneficial effect.

Statistics begin to reflect the trend of these hopeful developments, and notifications of all forms, despite better means of detection, fell from 52,600 in 1948 to 49,400 in 1950 and 1951.

There has thus been an acceleration in the marked progress which was made in combating the disease between the world wars, when deaths fell from about 58,900 in 1918 to about 25,600 in 1939.

Wartime conditions inevitably conduce to an increase in tuberculosis, and in 1940 and again in 1941 the number of deaths rose to over 28,000. This increase, however, was checked, and the figures for 1942, 1943 and 1944 were approximately 25,500, 25,600 and 24,100 respectively. The fall continued in 1945 and 1946 and a slight rise in 1947 was followed by further progressive falls.

These mortality figures are the most reliable criterion of the trend of tuberculosis, as against the number of notifications of the disease which are not necessarily an accurate reflection of its incidence. In 1918 the total number of formal notifications was about 90,000; in the pre-war year 1938 it was about 50,000; and since then it has fallen to 49,000 in 1951 after a rise to a highest figure of 54,000 in 1943 and 1944.

The relation between respiratory and non-respiratory types of tuberculosis, which hitherto has shown no substantial deviation from year to year (although such a factor as increasing pasteurisation of milk may materially affect the extent of non-respiratory tuberculosis as time goes on), may be gauged from the fact that in 1950 and 1951 of the total 15,970 and 13,806 deaths, 14,083 and 12,031 respectively were respiratory cases.

Treatment

The rate of admission of patients to tuberculosis institutions for in-patient treatment is affected by the nursing shortage, but has become a less serious problem as nursing resources have been steadily augmented and other efforts made to make additional beds available.

At the end of December 1951 there was an increase of 866 full-time and 187 part-time nurses since January 1950.

The waiting-list at the end of 1951 was about 7,000 against about 10,000 a year earlier, and the length of time for which patients have to wait for beds has generally been much shortened.

In order to reduce the waiting period for beds for tuberculosis patients, certain recommendations, based on advice from the Tuberculosis Standing Advisory Committee, were sent to all hospital authorities and local health authorities. These were:

1. Beds to be allocated for tuberculosis cases in general hospitals.
2. Where practicable, home treatment to be combined with short periods as an in-patient.
3. Importance of chest clinics linking energetic and preventive and after-care activities with clinical work.
4. Utilising the full resources of mass radiography as a means for finding latent cases of infection.
5. Suitable occupational resettlement as part of after-care arrangements.

The number of additional beds for tuberculosis treatment made available between the middle of 1948 and the end of 1951 was about 6,000.

Mass Radiography

The introduction in 1943 of mass miniature radiography for group examination to detect respiratory tuberculosis in its earlier stages was an important advance. By 1951, 56 units were in operation in England and Wales, and the Ministry itself operates one at a fixed centre in London as a training and research unit. They are examining about 2 million people a year, a large proportion of whom are industrial workers of every kind. In all, some 8 million persons have been examined to date, about 95 per cent. of whom were found to be normal at the time of examination. The number of cases in which clinical investigation resulting from evidence of abnormality in the miniature film led to diagnosis of active tuberculous conditions was approximately 28,000, or about 3·5 per thousand of all the persons examined.

New Drugs

Since the war, two new drugs, streptomycin and para-amino salicylic acid, have come into the picture. Para-amino salicylic acid (P.A.S.) alone has been shown clearly to be beneficial in the treatment of acute progressive tuberculosis. It seems, however, to be less effective than the streptomycin alone. The combination of the two drugs has a most marked effect in delaying or preventing the emergence of streptomycin-resistant variants of the infecting organisms. It is not yet possible to assess the effects of prolonged P.A.S. or combined treatment. The main object of this treatment is to prepare selected cases for collapse treatment or chest surgery. At the same time the combination enables streptomycin to be effectively administered for longer periods and for a wider range of lesions. Neither streptomycin nor P.A.S. is free from toxicity. In the present state of knowledge the prophylactic use of P.A.S. is not justified. It is, moreover, a costly drug.

The favourable reports from America suggest that isonicotinic hydrazide may represent a considerable advance in the chemotherapy of tuberculosis. This drug, therefore, certainly needs to be fully investigated; but even on present evidence, it is clear that it would be unwise to use it indiscriminately, for animal experiments indicate the possibility of toxicity, particularly when

the drug is used for a considerable time. There is the possibility of developing resistant strains of tubercle bacilli in the community, for such strains can develop in the laboratory.

It would seem prudent for the time being to limit its use to the treatment of in-patients in such hospitals as are equipped with a full laboratory service.

B.C.G. Vaccination

In 1949 the then Minister of Health, acting on expert medical advice, made arrangements for the use in this country, within certain limitations and under controlled conditions, of the vaccine known as B.C.G. (*Bacillus Calmette-Guerin*).

It is not yet certain that B.C.G. vaccination gives complete immunity against tuberculosis, and only experience will show how far it is effective under conditions in this country. But there is little doubt that at least it reduces the risk, and there is also some evidence that if a vaccinated person does subsequently contract tuberculosis the vaccination is likely to make it less severe.

The scheme for using B.C.G. in this country is in two main parts. Vaccination is being offered, subject to certain individual medical tests, firstly to all hospital nurses and medical students as a routine precautionary inoculation, and secondly to children or other persons known to be in close contact with a T.B. sufferer, if approved by a chest physician.

Changes in the Tuberculosis Service

Under the National Health Service Act, 1946, responsibility for insstitutional and out-patient treatment, including mass radiography, passed on July 5, 1948—the date of the operation of the National Health Service—from County and County Borough Councils to the Regional Hospital Boards. But the local authorities remain responsible, as the Local Health Authorities under the Act, for the preventive care and after-care work, and their arrangements over such matters as home visitation by tuberculosis health visitors working in association with the local Chest Clinic, provision in case of need of such requisites as bedding or extra nourishment, boarding out of child contacts, co-operation with the Ministry of Labour and National Service officials in the occupational resettlement of tuberculous persons, facilities for domiciliary occupational therapy, and so on.

Under the National Assistance Act, those who suffer loss of income undergoing treatment for respiratory tuberculosis can obtain additional assistance from the National Assistance Board. The main object is to afford a reasonable standard of maintenance for a patient and his dependents during the period of treatment and so encourage the sufferer, both for his own and the community's interest, to give up work in favour of early treatment although he is not at that stage incapacitated.

PUBLIC HEALTH IN 1950
CHIEF MEDICAL OFFICER'S REVIEW

Sir John Charles, Chief Medical Officer of the Ministry of Health, looks back over the first half of this century in his report* on the state of public health in England and Wales published in July.

* Report of the Ministry of Health, Part II, on the State of the Public Health, being the Annual Report of the Chief Medical Officer for the year 1950, Cmd. 8582. H.M.S.O., price 6s.

Concerning tuberculosis he makes the following observations:

Compared with 1900 the death rate from respiratory tuberculosis is less than a quarter of what it was. For the non-respiratory forms of the disease the reduction is even greater, and in 1950 the death rate was less than one-thirteenth of the mean annual death rate for 1891-1900. " These simple statements conceal half a century of endeavour and achievement," points out Sir John. " The battle is not yet over but the outcome is certain." But this " brilliant picture of progress " has its darker aspects. Cancer, and especially cancer of the lung, remains the greatest problem of medicine, while some diseases have noticeably increased, such as poliomyelitis and coronary thrombosis. Less serious blemishes are the prevalence of a mild form of dysentery, food poisoning, and the continued defiance of the common cold.

About the year 1950 itself, Sir John says that the remarkable fall of 20 per cent. in the number of deaths from tuberculosis compared with 1949, when the number was the lowest then recorded in this country, should greatly encourage us, adding: " But much remains to be done, and a plea is made that the problem should be viewed as a whole, avoiding over-emphasis of certain features and under-estimation of others. Prevention of an admittedly preventable disease should not be beyond our powers given close integration of the clinical, preventive and social side of the work."

During 1950 the number of mass miniature radiography units working among the civilian population was 53, 10 more than in the previous year. The total number of examinations since October 1943 was a little short of 6 million, and more than 30,000 examinations per week were being made throughout 1950. Of the total examined, some 95 per cent. were found to have no abnormal chest condition. Previously unsuspected active tuberculosis of the lung was revealed in less than 4 per 1,000 persons examined, but was the cause of more than 13 per cent. of the total notifications of respiratory tuberculosis. Steady progress was made with B.C.G. vaccination. It is estimated that up to the end of the year more than 20,000 persons had been vaccinated, of whom about one-third were hospital nurses and medical students.

MINISTRY OF HEALTH

T.B. Deaths Lowest on Record—Less Waiting Time for Treatment

Deaths from T.B. in England and Wales last year were the lowest ever recorded. This was announced in London on July 9 by the Minister of Health, Mr. Iain Macleod, when he addressed the third Commonwealth Health and Tuberculosis Conference organised by the National Association for the Prevention of Tuberculosis.

But the Minister warned that the progress now being recorded in the fight against this disease gave no ground for complacency or slackening of effort.

Mr. Macleod recalled that it was about the start of the century that organised action against this scourge first began to be seriously considered.

" From then onwards until today, with the resources of the National Health Service now arrayed against the disease, there has been almost unbroken progress," he added. " And what makes the last three years significant is the evidence they have shown that the rate of progress is becoming faster—a most promising sign.

" In the first decade of the century in England and Wales over 1,100 people per million of population died of respiratory tuberculosis every year. Last

year the number was 275 per million. In the last twenty years the number of tuberculosis deaths has been brought down by 60 per cent., although the population has meanwhile increased by a tenth. The toll of the disease is still heavy, but the progress has plainly been great, and the pace of it is becoming quicker.

" In 1949 the tuberculosis deaths were 2,000 fewer than in 1948, for which the figure was the lowest on record. In 1950 there was a yet more notable fall, to a figure 4,000 fewer than in 1949; and provisional figures for 1951 show another drop of some 2,000. This means that last year about 8,000 fewer people died from tuberculosis than in 1948—a reduction of over one-third—which is by far the biggest gain in any three-year period since records began.

"Notifications of new cases are also now slightly falling. In the circumstances this suggests that along with the much decreased mortality we are also making headway against the attack rate of tuberculosis."

When the Conference was last held in 1949 the most serious post-war problem in the fight against T.B. in this country was at its worst. The list of patients awaiting admission to in-patient treatment, mainly due to the great shortage of nurses, was at its peak of about 11,000 in England and Wales. It had now been brought down to about 7,000. Even more important was the fact that the waiting period for a bed had for the majority of patients in almost every part of the country been much shortened—very often to a few weeks instead of several months and to only a few days where there was special urgency.

Since the National Health Service began over 6,000 more beds had been made available for T.B. treatment, nearly two-thirds of them in the last two years. There was, unfortunately, still a good deal of difficulty about patients recommended for thoracic surgery for which present resources were limited. The problem of establishing and staffing extra surgical units of this kind was not one which could be quickly solved.

Mass miniature radiography now had an established place in the armoury against respiratory T.B. At the end of 1948 there were 36 units at work in England and Wales and they had examined about 3,000,000 persons. By the end of last year 57 units were operating and the number of persons examined totalled about 8,000,000. Of these roughly 28,000—or between 3 and 4 in every thousand—were found to have an active tuberculous condition.

B.C.G. Vaccination

Referring to B.C.G. vaccination, Mr. Macleod said: " It is not yet felt that the time has come to make the vaccine available for general or indiscriminate use among the population at large. This view, though it may be thought unduly restrictive by some, is based on responsible expert judgment, and is not due to limitations of supply of the vaccine or to financial economy. Neither is it necessarily a fixed policy. My Ministry will take careful account of whatever expert advice may be forthcoming in the light of continuing study and research."

Mr. Macleod also made a plea for united action by the various branches of the Health Service in the field of T.B. prevention and after-care. " In this co-ordination the Medical Officer of Health holds the key position and we look to all the others concerned to give him every help," he said.

TRENDS IN THE TREATMENT OF TUBERCULOSIS AT TORONTO

THE following brief extracts from an interesting paper on recent trends in the treatment of tuberculosis prepared in March 1952 by Drs. H. S. Coulthard, K. F. Davis, D. R. Garrett, H. M. Ross and G. A. Wicks appeared in the 1951 Annual Report of the Toronto Hospital.

General Remarks upon Sanatorium Routine and Bed Rest

Although sanatorium routine and bed rest do not constitute " recent trends " in the treatment of tuberculosis, some mention should be made of the value of such measures. Rest in bed in the recumbent position if properly followed will diminish the stretch upon areas of tuberculosis in the lung. Thus relaxed, and with relative immobilisation, diseased areas and surface of granulation tissue are apposed. This immobilisation tends to prevent the stretching of delicate connective tissue which gradually replaces active tuberculous disease and favours " healing " by fibrosis. Collapse therapy, chemotherapy, immobilising operations and resections are supplementary to sanatorium routine to assist the host in mustering a favourable cellular, biological or immunological reaction to the disease processes.

General Trends in Collapse Therapy

(*a*) To an increasing degree we have refrained from instituting artificial pneumothorax, and have relied upon bed rest with or without chemotherapy and with or without artificial pneumoperitoneum, followed by thoracoplasty or resection when indicated. The main indication for thoracoplasty is, of course, residual cavitation in the upper lung area, but when the tuberculous lesion is solid, resection is now being used to an ever-increasing extent.

(*b*) Localised areas of caseation which do not show evidence of clearing or excavating after a reasonable period of bed rest are now being considered for some type of resection.

(*c*) Extra-pleural pneumothorax has not been applied here within the past six years, but we are employing apicolysis in conjunction with thoracoplasty in an increasing number of suitable cases for collapse of tuberculous disease in the extreme apex.

(*d*) Frequently, in recent years, surgical collapse by thoracoplasty is being advised before discharge, to consolidate gains made by a period of bed rest for a moderately advanced lesion which, on bed rest, has partially cleared and become partially fibrosed, even with no evidence of residual cavitation. This trend is the result of experience in which we find that this type of lesion without collapse treatment so frequently becomes reactivated following the patient's discharge from sanatorium. It may be that local resection is equally applicable in some of these cases.

At this sanatorium no collapse therapy or resection has been undertaken recently without a recent examination of the bronchial tree by bronchoscopy. Resection through the site of actual tuberculous bronchitis is never carried out for fear of a broncho-pleural fistula developing. Artificial pneumothorax is is not attempted in the presence of a moderate degree of tuberculous bronchitis. Thoracoplasty is postponed if marked tuberculous bronchitis is found and streptomycin with PAS is given.

Present Attitude regarding Streptomycin Therapy

For the past two years our aim has been to give concurrent oral PAS to all patients who are receiving streptomycin intramuscularly. During the past year, with the likelihood of bacterial resistance minimised by the use of intermittent streptomycin and continued PAS, we have increased the duration of antibiotic treatment. Care is taken to co-ordinate the use of streptomycin and PAS with the general plan of treatment for the individual patient, and except in a long-term treatment course we do not use them routinely, but only in distinctly sub-standard risks. We do, however, use streptomycin and PAS with all pulmonary resections, decortications and with all operations at which any tuberculous tissue is opened or disturbed.

We feel that streptomycin with PAS has, in brief, fulfilled the following functions:

(*a*) Halted exudative, extending parenchymal disease and led to varying degrees of resorption.
(*b*) Reduced the overall risk of resection operations and rendered suitable for such operations those patients who formerly would not have been considered for resection.
(*c*) Contributed to the control of post-operative spread of tuberculosis following surgery.
(*d*) Favourably influenced the course of tuberculous bronchitis, and thus has permitted many patients with this complication to receive later collapse therapy.

Dosage and Type of Streptomycin Used

Actually, dosage of streptomycin is governed by the nature of the tuberculous disease, the size of the patient, the purpose of treatment, etc. Since July 1, 1951, in the majority of cases of tuberculosis except tuberculous meningitis or miliary tuberculosis in adults we have been giving 1 gramme of streptomycin (calcium chloride complex) intramuscularly in one dose twice weekly with a daily oral dose of 10 grammes of PAS or its equivalent. This regimen has been continued for at least six to nine months.

During the past year there has been a definite tendency to increase the total period during which streptomycin therapy is applied. In the majority of cases the present duration of streptomycin treatment at this hospital is approximately six months to a year.

For those patients undergoing pulmonary resection or representing a distinctly sub-standard risk for thoracoplasty we now usually give a three-month course of streptomycin in twice-weekly doses commencing a few days prior to operation. The same routine applies to nephrectomies for tuberculosis and for any infection in which tuberculous tissue is opened or disturbed.

Para-aminosalicylic Acid

(*a*) *In conjunction with streptomycin.* Since March 1948 we have been using para-aminosalicylic acid orally with streptomycin intramuscularly. It was apparent that " combined " therapy in certain dosages had the following advantages over the use of stretomycin alone:

(i) Marked delay in the appearance of tubercle bacilli resistant to streptomycin.
(ii) Enhanced clinical results.

When the advantages of this "combined" therapy were evident, we began "covering" all streptomycin administered intramuscularly with PAS orally in doses of 10 grammes daily.

(*b*) *Alone.* During the past year our use of PAS alone has almost ceased and only a few patients are now receiving PAS alone.

Artificial Pneumothorax

The number of cases in which artificial pneumothorax treatment was instituted has markedly but not steadily decreased, owing to a wider use of artificial pneumoperitoneum, thoracoplasty and resection.

Pneumolysis

If artificial pneumothorax requiring pneumolysis is going to endanger the outcome, we feel that the pneumothorax should be abandoned and replaced by thoracoplasty or the lesion should be treated by resection.

Pneumoperitoneum

This has been applied, almost never with phrenic paralysis and with or without streptomycin, with increasing frequency with the following indications:

(*a*) In preference to artificial pneumothorax to avoid complications.
(*b*) As supportive and preparatory treatment for thoracoplasty or resection.
(*c*) For cases in which it is safer than unilateral or bilateral pneumothorax.
(*d*) For patients where artificial pneumothorax is indicated but is contra-selective.
(*e*) To assist in obliterating pleural spaces.

Phrenic Paralysis

Phrenic paralysis is used here very much less frequently than before, the main reasons being the limited duration of benefit in the case of temporary paralysis, the danger of retention of secretions in the lower lobe lesions, and the undesirability of having a paralysed diaphragm to contend with at and following a thoracoplasty operation or a lobectomy or lesser resection.

Thoracoplasty

This procedure has proved to be the most widely acceptable surgical procedure in use at Weston in recent years. The mortality and morbidity rates are extremely low and the sputum conversion rate is about 85 per cent. The operation is reserved for stable disease and is recommended for persistent cavitation in the upper lobe, or for permanent collapse of areas of lung which have been extensively destroyed and fibrosed. Very recently there has been a trend towards treating some of these cases by resection, because of the solid nature of the lesions.

Thoracoplasty operations vary in technique and extent and often are associated with apicolysis when this procedure offers better collapse of the lesion. For obliteration of empyema spaces, thoracoplasty is combined with parietal pleurectomy in order to obtain the necessary collapse. Thoracoplasty may also be used to obliterate dead space following pneumonectomy or lobectomy, when the remaining lung tissue is known to have been the seat of recently active tuberculosis.

Pulmonary Resection

From 3 to 9 cases a year in 1941 to 26 cases in 1951 have received this form of treatment. The indications for this operation are still growing.

Present practice demands the use of systemic streptomycin to cover all cases of resection. This has decreased the incidence of empyema and leakage from the bronchial stump with mediastinitis and broncho-pleural fistula, and has been valuable in the control of post-operative spread of tuberculosis.

Post-resection thoracoplasty has gradually assumed a larger place, until at present our rule is to do this operation some weeks after resection in those who have known disease, or those who have suffered an exacerbation of disease, in the remaining lobe or lung.

It has become our practice in a weekly clinical-pathological conference arranged by the pathologist to review all resected specimens as to their gross and histological appearance, and to correlate this with the serial X-rays and clinical course prior to the operation. This has led us already to modify some of our opinions, and occasionally alter previously accepted criteria, both for resection and for collapse therapy.

WORLD HEALTH ORGANISATION

The Director-General of the World Health Organisation has sent us the Annual Report on the work of his organisation. It covers a wide field of activities, of which the section on tuberculosis is of particular interest to readers of this Journal. In co-operation with the United Nations Children's Emergency Fund (UNICEF) it has taken over and expanded B.C.G. campaigns in Ceylon, Ecuador, Egypt, India, Pakistan and the Philippines and established tuberculosis control and teaching centres in Burma, Ecuador, India, Pakistan and Thailand. It has aided control projects, usually including B.C.G. vaccination, in China, El Salvador, Finland, Hong Kong, Jamaica, Malaya, Singapore and Yugo-Slavia. In Austria diagnostic laboratories have been set up and central dispensaries reorganised, and supplies have been furnished for the treatment and control of tuberculous meningitis. The control programmes in Greece and Italy have been extended and in Turkey the training centre has been aided.

ANNUAL REPORT OF THE CITY OF DURBAN MEDICAL OFFICER OF HEALTH

We have received the Annual Report of the Medical Officer of Health for Durban, South Africa, for the year ending June 30, 1951. The preliminary census figures for 1951 for Durban show a total population of 424,160, made up of European, native, coloured and Asiatic peoples—viz., European, 129,380, Coloured, 14,958, Native, 134,451 and Asiatic 145,371.

The number of known cases of tuberculosis—all forms—comprises 6,978, made up of 1,164 European, 503 Coloured, 3,508 Native and 1,803 Asiatic. The Medical Officer makes the following interesting observations on local factors:

The low prevalence of tuberculosis among Durban Europeans rules out the sub-tropical climate with its trying heat and humidity in summer as a factor in the etiology of tuberculosis locally. The basic environmental factor is urbanisation of the non-European races, who suffer heavily in the process

of adjustment from conditions of their primitive origin to those of a modern industrial city. Overcrowding, fatigue, lack of rest, poor feeding, lack of recreation and a low living standard generally, combine to create conditions ultra-favourable to the spread of tuberculosis. Important contributory factors are the belated discovery of " open " cases and the rapidity with which the minimum stage of pulmonary lesions is succeeded by the " open " or infectious stage. In this connection, the shortage of bed-space and the reluctance of Natives particularly to remain in hospital are to be noted as important.

The problem of tuberculosis among non-Europeans is largely a socio-economic problem, soluble in terms of improvement in their living environment, their advance to a higher earning capacity and higher standards of living—better housing, nutrition, education and recreation.

Medical aspects of the problem relate:

(*a*) The progress of primitive peoples toward a higher level immunisation against the infection.

(*b*) Improved case-finding as a precursor to:

(i) efficient isolation of " spreaders ";

(ii) removal of cases in the early stages from work and fatigue.

NOTES AND NOTICES

BRITISH STUDENT TUBERCULOSIS FOUNDATION

Student Rehabilitation Unit, Pinewood Hospital, Berkshire

We have recently received a communication from which the following quotation is taken from Dr. N. Malleson, hon. secretary of the Foundation, 6, Gordon Square, W.C.1. It is of particular interest in relation to Dr. Sickenga's contribution on the Dutch Students' Sanatorium in this issue.

" The British Student Tuberculosis Foundation was started over six months ago. It aims at opening in this country a 100-bedded rehabilitation centre where students convalescing from tuberculosis can be fitted for return to college by courses of study, including lectures, set reading and essay writing, tutorials, etc. In a little over eighteen months British students have collected some £20,000 towards a fund for this purpose.

" This April the North-west Metropolitan Regional Hospital Board agreed to open a small experimental unit for men at Pinewood Hospital, a sanatorium situated in open heathland near Wokingham in Berkshire. This centre is expected to be ready in September; it has accommodation for sixteen student patients in one self-contained ward. This is to be divided into three parts: a dining-room where all meals will be taken, a lounge, and a bed-ward where students can read in silence during rest periods. The medical care and supervision of the patients will be the responsibility of the physician superintending the hospital. The academic side will be the responsibility of the Foundation. Those admitted will have to be recovering satisfactorily; they must be non-infectious in the sense of sputum-negative. As only a minimum of nursing staff will be available, students must be fit to do a few light jobs for themselves, such as making their beds. They must be able to take all meals up and stay up and dress for some four hours a day at least. Patients will be expected to abide by the rules of the sanatorium during their three-four month stay there.

" The Foundation is to be responsible for occupational treatment, and is now making arrangements for University teachers, primarily from the University of London, to visit the centre regularly to give lectures and to supervise individual courses of study.

" Entry is open to full-time male students from all parts of the British Isles of any race, colour or creed. The Foundation will be pleased to receive, at the earliest opportunity, applications from any full-time student who would like to come to Pinewood and who expects to be fit to do so by the autumn or at any time throughout the coming year. Application forms and full details will be supplied on request."

N.A.P.T.

Third Commonwealth Health and Tuberculosis Conference

The third Commonwealth Health and Tuberculosis Conference organised by the National Association for the Prevention of Tuberculosis was held at the Central Hall, Westminster, from July 8 to 11, inclusive. Over eight hundred representatives from fifty-one countries were present: doctors, hospital administrators, public health officials, nurses, social workers and private

individuals. Although the Conference was specially concerned with tuberculosis in the Commonwealth, the control of the disease is a world-wide problem. It is therefore of vital importance that knowledge and experience should be shared, so it was particularly gratifying that so many foremost authorities from other countries were present. A detailed report of the proceedings of the Conference will be published by the N.A.P.T.

POST-GRADUATE COURSE IN TUBERCULOSIS AND DISEASES OF THE CHEST

(Province of Quebec, Canada)

The Third Post-graduate Course in Diseases of the Chest, sponsored by the Provincial Committee for the Prevention of Tuberculosis, was held in Montreal in April. Well over two hundred physicians, nurses and other workers interested in tuberculosis registered for the course.

The Committee's sub-committee, responsible for planning the course, consisted of Dr. Fernand Hébert, Medical Director of Hôpital du Sacre-Cœur, Cartierville, P.Q.; Dr. Alphonse L'Espérance, Medical Director, Hôpital Laval, Quebec; and Dr. B. G. Bégin, a member of the surgical staff of Dr. Hébert's hospital. This sub-committee succeeded in making unusually fine physical arrangements for the course, and in gathering together a remarkable array of talent.

Sessions were held at both the University of Montreal and McGill University as well as at three of the sanatoria on the island of Montreal. The Deans of the Faculties of Medicine of the Universities of Montreal and McGill presided at two sessions. All of the speakers, and all who participated in the discussions, were encouraged to speak in the tongue—either French or English—with which he or she was most familiar. These efforts to make the sessions truly bilingual added zest to the discussions and proved to be popular all round.

The emphasis in this year's course was placed on the presentation, and the discussion, of subjects of special interest to physicians and others working in health units, chest clinics, sanatoria and tuberculosis hospitals. The subjects discussed included socio-economic factors favouring tuberculosis control; the prevention of tuberculosis in personnel employed in sanatoria; the differential diagnosis of pulmonary lesions of minimal extent; the treatment of minimal pulmonary tuberculosis; the status of antibiotic therapy in tuberculosis; broncho-pulmonary segments and their significance; anæsthesia in thoracic surgery; tumours of the lungs, both benign and malignant. Several speakers from the United States of America participated in the programme. Among these were: Dr. Julia M. Jones of Bellevue Hospital, New York City; Dr. Nicholas D. D'Esopo of the Veterans' Administration Hospital, Sunmount, New York; and Dr. Chevalier L. Jackson of Philadelphia, Pennsylvania, at present the President of the American College of Chest Physicians.

The Honourable Dr. Albini Paquette, Minister of Health for Quebec, was the principal guest and special speaker at a banquet before the closing of the conference.

ANNUAL MEETING OF THE CANADIAN TUBERCULOSIS ASSOCIATION

(REGINA, PROVINCE OF SASKATCHEWAN)

THE Canadian Tuberculosis Association held its 52nd Annual Meeting in Regina in mid-June. Representatives of all of the provinces, including Newfoundland, were present. In addition, the members of a Public Health Mission from India and Pakistan, led by Lieut.-Col. T. C. Puri, Deputy Director-General of Health Services, New Delhi; one of the outstanding leaders in tuberculosis work in the United States of America, Dr. W. H. Tucker, of Minneapolis, Minnesota; and a goodly number of prairie-province residents who are genuinely interested in the prevention and control of tuberculosis, attended the sessions.

The papers read at the Medical Sessions were for the most part of a high order. They included an account by Dr. G. E. Maddison of an outbreak of tuberculosis in a small town in New Brunswick; a preliminary report on functional studies before and after pneumonectomy by Dr. Fernand Gregoire of Montreal; an interesting presentation—in lantern slide form—of the correlation of the clinical and pathological findings in patients subjected to resection of a portion or all of one lung, by Drs. Lee and Ewart of Hamilton, Ontario; a paper entitled " Limited Thoracoplasty " by Dr. Sinclair of St. Vital, Manitoba; a contribution on pulmonary hydatid cysts by Dr. H. E. Meltzer of Edmonton, Alberta; and from Dr. Harrison, of Vancouver, B.C., an account of his own experience with pulmonary resection in tuberculosis.

AUSTRALASIAN ANNALS OF MEDICINE

WE have just received the first volume of this Journal of the Royal Australasian College of Physicians, and we congratulate the Editorial Committee on an admirable first issue which augurs well for the future.

The President of the Royal Australasian College of Physicians, Dr. A. W. Holmes à Court, makes the following observations in his Foreword:

" *Australasian Annals of Medicine* has been established as the official organ of the Royal Australasian College of Physicians and is to be published in May and November of each year.

" Since its inauguration in 1938, the College has contributed to the stature of medicine in Australia and New Zealand by establishing standards of higher education in medicine and by promoting medical research. In the judgment of its Council, the time has arrived for the publication of an Australasian journal devoted to internal medicine and the allied sciences.

This journal is intended to cover a wide field. It will not be restricted to the work of members of the College, but is designed as a medium for the presentation of original work carried out and investigations made in medicine and medical sciences in Australia and New Zealand."

As far as the contents are concerned, among the admirable articles of a high standard our readers will be interested in a comprehensive paper on " Vascular Abnormalities of the Lung," by Dr. Cyril Fortune, and one on " Dissecting Aneurysm of the Aorta confirmed by Angiocardiography," by Dr. E. J. Halliday.

The issue contains a brief history of the Royal Australasian College of Physicians and the Proceedings of the Ordinary Meeting of the College for 1951.

NEW YORK TUBERCULOSIS AND HEALTH ASSOCIATION "WHITHER TUBERCULOSIS"

We publish the following summary from a paper "Whither Tuberculosis," a statistical review of reports from selected American and European communities by G. J. Drolet and A. M. Lowell, published in *Diseases of the Chest*, May 1952.

Tuberculosis mortality has been declining at an accelerated rate, especially during the past few years. Between 1945 and 1950, the death rate fell in England and Wales by 36 per cent.; in the United States, 41 per cent.; in Canada, 43 per cent.; in France, 49 per cent.; in Finland, 50 per cent.; Italy, 53 per cent.; Norway, 55 per cent.; Denmark, 58 per cent.; Sweden, 63 per cent.; Iceland, 71 per cent.; and in the Netherlands, 78 per cent.

Tuberculosis morbidity, on the other hand, has tended to remain on a high level: 100,772 new cases were reported in the United States in 1940 and 121,228 in 1950; in Canada, 10,226 formerly and 12,429 latterly; in England and Wales, 49,967 notifications ten years ago and 52,062 last year; in Denmark, 2,687 new pulmonary cases in 1940 and 2,507 in 1950. Effective prevention of tuberculosis obviously remains elusive so far.

Improved forms of treatment in tuberculosis are responsible for the lowering of "community case fatality rates," which ranged between 50 and 60 per cent. in 1940, but were reduced in 1950 to 31 per cent. in England and Wales, 29 per cent. in Scotland and in Canada, 28 per cent. in the United States, and only 20 per cent. in Denmark.

Children have generally enjoyed greater reductions of tuberculosis mortality than older groups of the population. Their death rate in the United States, in spite of an increasing concentration of population in urban, congested centres and large Negro groups, has been steadily reduced. Among those under 51 years of age, in the year 1949 it averaged for the entire country 3·9 per 100,000. In the state of Minnesota, it was as low as 1·3. Abroad, the same year, the death rate of the children in Denmark was 3·6; but, in Norway, it was 8·1; and in England and Wales, 12·1.

B.C.G. vaccination has been widely used in the Scandinavian countries—little in Iceland—and only very recently to any extent in the United States or in England and Wales.

In the state of New Jersey, special measures for the protection of school children—periodic X-ray examination of school personnel along with tuberculin testing and X-raying of pupils annually—resulted in 1949 in reducing the death rate of children under 15 to 2·7, or only one-third of that prevailing in Norway. The death rate of the children in Denmark was 44 per cent. of that of Norway.

THE FIGHT AGAINST TUBERCULOSIS IN TURKEY

The first training course in tuberculosis control was set up by a WHO advisory team in Istanbul. Its objectives were:

1. To demonstrate modern methods of control in an area of high incidence.
2. To evaluate results achieved.
3. To train personnel from Turkey and other Eastern Mediterranean countries in prevention, case-finding and treatment.
4. To collaborate with and assist the Government of Turkey in its efforts to combat tuberculosis.

The Turkish Government and the Istanbul Antituberculosis League sponsored the construction of a modern building to house the Centre, thus facilitating the team's work.

The mortality rate in Turkey is about 200 per 100,000 as contrasted with 13 per 100,000 in Denmark, where the rate was comparable with Turkey's half a century ago. The morbidity rate is difficult to determine, but positive reactions among students were shown to be 91·2 per cent. and over varied population groups 70 per cent. Among 62,197 mass X-ray examinations 2,386 were " suspect," suggesting that 50 to 60 of every 1,000 apparently healthy subjects needed control examinations and 20 to 25 had active tuberculosis or needed medical care.

Training courses were given, in the space of 18 months, to 430 physicians and 460 nurses and other medical personnel. The medico-social problems were emphasised. The nurses were given practical experience, and the Istanbul Antituberculosis League which trains annually 10 nurses for special work in tuberculosis was reorganised. Lectures, films and exhibitions were arranged to reach the public, both in and out of Istanbul, the Centre there trying to serve as a model and " testing bench " for other areas.

The Government of Turkey is actively engaged in a fight against tuberculosis. To this end 22 new dispensaries are to be created in 1952 and a large-scale B.C.G. vaccination campaign is to be undertaken. The number of sanatorium beds was raised from 1,023 in 1948 to 4,107 in 1951, and the social security provisions are being improved and expanded. WHO's team has undoubtedly provided new stimulus as well as technical guidance for the fight.

TUBERCULOSIS IN THE PHILIPPINES

We have received an Analysis and Evaluation of Tuberculosis in the Philippines from the World Health Organisation. This report bristles with figures, some of which do not tally, but which make clear the dimensions of the problem, for, despite improvement in recent years, tuberculosis still leads the mortality figures and infects about one in seventeen of the population. Among nineteen and a half millions in 1949 and a total mortality of 231,151, tuberculosis accounted for 29,640 deaths, of which 27,815 were due to pulmonary disease. Tuberculin tests show that 50 per cent. of school children are infected before the age of eighteen, when the rate rises to 100 per cent.; this figure is already reached at the age of three by children in the slums of Manila.

The climate, the social conditions and habits of the people throw light on these figures. The excessive heat in the dry season and long months of heavy rain in the wet season keep overcrowded families in their huts. The Philippines are mainly agricultural, but large tracts are virtually uninhabited and untilled owing to insurgency, which swells the persistent townwards drift of the people by driving them into towns to seek safety in numbers. These unite with those already out of work in aggravating an acute housing shortage and the Government's main problem, unemployment. Malnutrition and beri-beri, together with the malaria endemic in some parts of the islands, sap resistance to infection; and this is encouraged by the widespread religious habit of kissing the images of saints and by the custom of the sharing of homes by two and three generations.

It can be seen that the medical aspect of the Filipino tuberculosis problem is inextricably mixed with the social, economic and educational conditions;

and the poverty of financial provision for control of the disease is harshly reflected in its ravages in plain terms of money.

There is only one public health laboratory in the Philippines. There are seven chest clinics with X-ray and laboratory facilities and two mobile X-ray units under the direction of the National Chest Centre, which also supervises tuberculin testing. The distance patients have to travel to clinics and hospitals is often prohibitive, and sanatorium beds are totally unequal to the demand. Voluntary organisations work with the official tuberculosis scheme; one, helped by a Government subsidy in the form of sweepstake funds, operates out-patient clinics and a sanatorium. There is no health insurance and little public assistance; the wage-earner of a family usually simply cannot afford to undergo treatment. An Act for the financial support of B.C.G. production and immunisation is, however, being contemplated, and a laboratory for the purpose will soon be set up. A new scheme for specialisation in tuberculosis by physicians and technicians is under way, the National Chest Centre at present being the only place for specialised training.

"ZEITSCHRIFT FÜR TUBERKULOSE"

We are informed that the 100th volume of the *Zeitschrift für Tuberkulose* is due for publication this year. In 1900 the original issue was the first German periodical of this speciality, and its contributors since have included Romberg, Sauerbruch, Pirquet, etc. The present volume surveys the development of the Journal from the outset and sets forth the contemporary state of research and practice, so that the issue is of practical value to both physicians and surgeons.

IN MEMORIAM

Sir Percival Horton-Smith-Hartley, C.V.O., M.A., M.D., F.R.C.P.

Consulting Physician to St. Barthomolew's Hospital and to the Brompton Hospital

The death of Sir Percival Horton-Smith-Hartley on June 30 last, within the walls of the great hospital in which he had been a student, marks the passing of yet one more of those physicians of the old school to whom the science and art of Medicine formed but a part of that general learning and broad intellectual culture which were the hall-mark of their life and character. Educated at Marlborough and St. John's College, Cambridge, he graduated with distinction, having been placed in the first class in both parts of the Natural Science Tripos. In 1891, still fresh in the Baccalaureate of his Alma Mater, he was elected to a Fellowship of his own College. Such initial honours were an almost certain indication of future eminence, and after study at St. Bartholomew's and also on the Continent he qualified with the least possible delay, obtaining the Conjoint Diploma, the M.B. Cambridge, and the Membership of the Royal College of Physicians in the same year, 1893. Three years later he received the Doctorate of his University, and in 1899, only six years after qualification, he was made a Fellow of the College of Physicians, and was chosen to give the Goulstonian lectures in the following year. After a few years as Assistant Physician to the Metropolitan Hospital, he was elected to the staff of St. Bartholomew's, and also to the staff of the Brompton Hospital. The study of chest disease soon became a major interest, and eventually constituted the chief part of his professional work, both in hospital and in private practice.

Not long after qualification, the writer had the privilege of spending three months in post-graduate study at St. Bartholomew's. One of his enduring recollections of those days is the figure of young Dr. Horton-Smith-Hartley, the junior Assistant Physician not long elected, with his fresh almost boyish face and his tireless energy. Some years later, when he was welcomed by Sir Percival as a colleague on the Brompton staff, that freshness, despite the passage of time, was still in evidence. As Senior Physician and Chairman of the Brompton Medical Committee, he ruled us with a rod of iron, with tenacious insistence on that strict adherence to precedent which characterised so many of the leading consultants of his time. Some of his younger colleagues were apt at times to feel some resentment, but to those whose judgment was fair and impartial it was nevertheless evident that his attitude was inspired mainly by his genuine love of the hospital, and his apprehension of the least possibility of its ancient standards suffering loss. His after-dinner speeches at the annual staff dinners portrayed the general culture and scholarship of the man. Like all really good orators, he prepared his speech beforehand with meticulous care, but its delivery gave the impression of easy grace, and of an effortless flow of poetic learning, as though from the very fount of Hippocrene.

Hartley's medical work was invariably careful and scrupulously accurate, as is abundantly evident in the textbook of Diseases of the Lungs and Pleuræ, of which he published six editions in collaboration with Sir Richard Douglas Powell, and also in his statistical researches on pulmonary tuberculosis published by the Medical Research Council. His greatest service to his profession, however, is, in our judgment, to be found in his contributions to general scholarship and in his maintenance of high general standards of culture in medicine, apart from its purely technical aspects. For this, especially in these degenerate days, should he be honoured; for this should his memory serve as a great example.

"Hæc olim meminisse juvabit."

M. D.

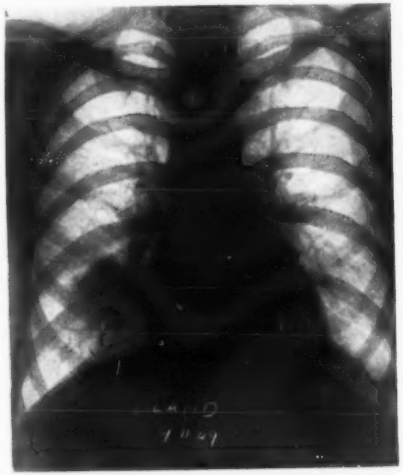

FIG. 1.

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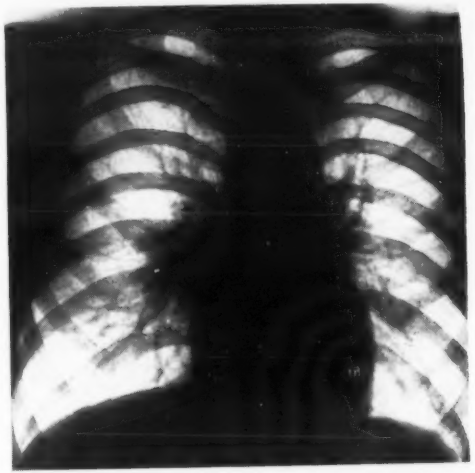
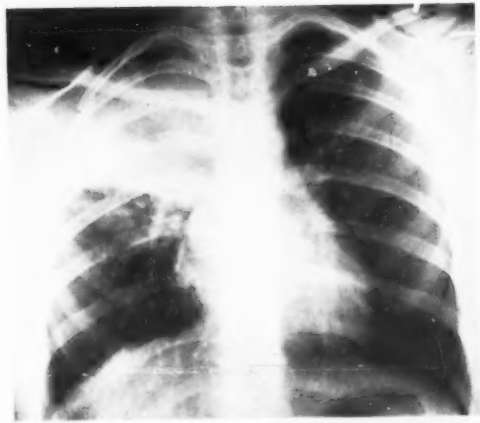


FIG. 3.

PLATE XXXI

FIG. 4.



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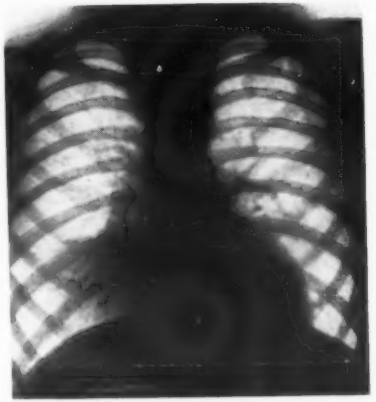
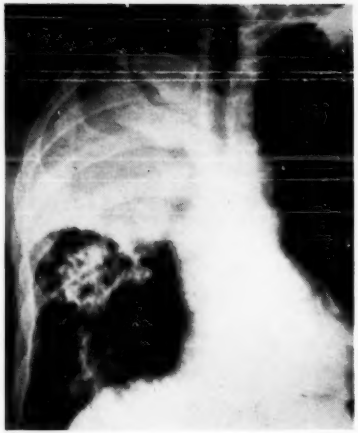


FIG. 6.

To face p. 240



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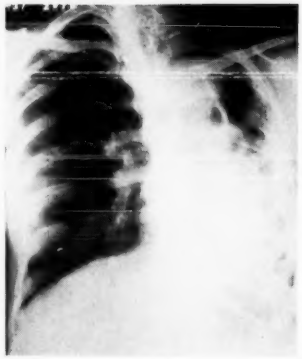

FIG. 8.

FIG. 9.

PLATE XXXII

FIG. 10.

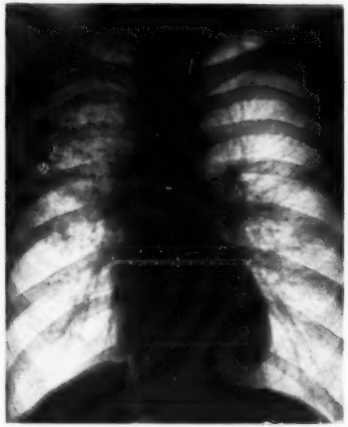

FIG. 11.

FIG. 12.

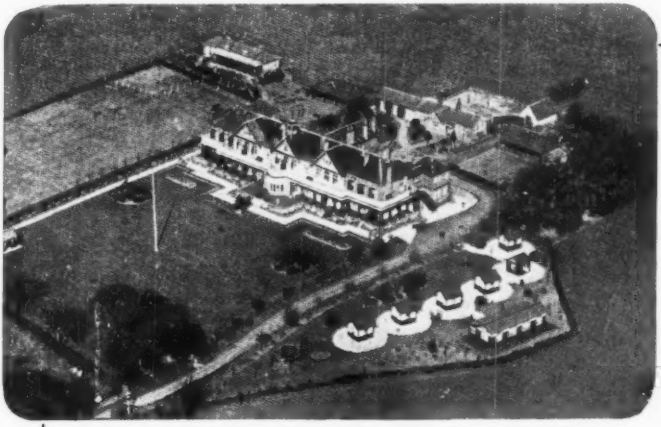

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Fig. 12.

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Fig. 11.

NAPT

NON-PULMONARY TUBERCULOSIS OF BOVINE ORIGIN IN GREAT BRITAIN

by

G. S. Wilson, M.D., F.R.C.P., M.R.C.S., D.P.H.
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Professor in Pathology, St. Bartholomew's Hospital Medical College, London.

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Fig. 10.

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